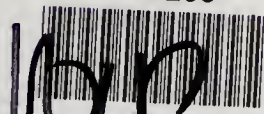


Sixteenth Edition

HUTCHISON'S CLINICAL METHODS

Bomford, Mason & Swash

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Hutchison's Clinical Methods

SIXTEENTH EDITION

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PREFACE TO THE SIXTEENTH EDITION

In its 78th year *Clinical Methods* has commended Balint, mentioned Weed, and gone metric and S.I. Modern students will be quite at home with the new units of measurement, however much the elderly clinicians, having wrestled with milliequivalents, may find themselves troubled by moles.

With this edition the book has lost the services of Donald Hunter—one of the best known and loved medical teachers of his day, who has presided over its destinies since Sir Robert Hutchison retired—and gained those of Stuart Mason and Michael Swash.

For this, its sixteenth edition, the book has undergone a very thorough revision. The first and last chapters are new; Michael Swash has virtually rewritten the chapter on the nervous system, and Frank Marsh has done the same to much of the chapter on the urine. A proper revision of the rest of the book would have been impossible without the generous help of many friends. In particular, we would like to thank Lesley Bidstrup, Sidney Crown, Harry Currey, George Dick, Eric Dunlop, Paul Grob, Peter Hall, John Hartgill, David Hughes, Brian Houghton, Anthony Jackson, David Jarvis, Barry Jay, George Jenkins, Greta Keenan, Alistair MacDonald, Alistair Mason, Harry May, Andrew Morrison, Joe Pegum, John Perrin, James Swallow, John Hermon Taylor, Gerald Tresidder, John Wright, Ian Wylie and a group of our students at the London Hospital. We are greatly indebted to all our colleagues but, nonetheless, accept full responsibility for any errors or omissions.

What to retain and what to delete has proved difficult. For example, mention of diphtheria, now an unimportant disease in the United Kingdom, might have been deleted. It has been retained, however, because the book has had a considerable circulation abroad, particularly in the developing countries. It has been translated, with permission, into Spanish; without permission it has appeared in two countries that do not acknowledge copyright. On the other hand, the short chapter on metabolic disturbance has been deleted because *Clinical Methods* is concerned with the doctor's relationship with his

patient during history taking and clinical examination, and not with management or treatment. It seemed to us, therefore, that this chapter fell outside our scope.

We hope that *Hutchison's Clinical Methods*, concentrating as it does on the methods of clinical examination, will continue to be useful to students and practitioners everywhere.

September 1974

Richard Bomford
Stuart Mason
Michael Swash

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1

CASE TAKING

The History — Routine Questions — The Physical Examination — Writing Out the History and Examination — Presenting a Case — Interpretation

Those who talk about the principles of medicine may be hard put if asked to enunciate them. 'Diagnosis should precede treatment (but not always)' is perhaps the nearest one can get. But diagnosis should precede treatment whenever possible.

There are two steps in the making of a diagnosis. The first is *observation* by means of clinical methods—history taking, physical examination and ancillary investigations—which are what this book is about. The second is *interpretation* of the information obtained, first in terms of abnormality of structure and/or function and then in terms of pathology. Interpretation in terms of pathology is largely outside the scope of this book and must be learned by practice, aided by textbooks of medicine and pathology. One should note that pathology means the science or study of disease. The modern habit of using this word as if it were synonymous with physical abnormality, e.g. 'I find no pathology', obscures clear thinking and communication.

THE HISTORY

The aim is to get from the person concerned an accurate account of his complaint and to see this against the background of his life as a whole. It is usual to record the findings under some or all of the following headings:

1. Presenting complaint
2. History of present illness
3. Previous history of illness
4. Menstrual history
5. Treatment history
6. Family history
7. Social and occupational history
8. Psychiatric history

However, in taking the history it is neither possible nor desirable to tie a patient down to such a sequence. He must be allowed to tell his story

in his own way. Further, it has been said that a good doctor begins the examination of a patient as the latter walks into the room—his general appearance, the way he walks, the way he answers questions and so on—and only finishes taking the history when the consultation is over. Occasionally a vital piece of information may come out just when the patient is leaving.

The list of headings may appear formidable and it does take some experience to know in a given case which part of the history is particularly worth pursuing. If, for instance, the patient's complaint is undue bleeding, a careful *family history* may virtually make the diagnosis. If he has chest symptoms, the fact that he worked with asbestos even twenty years earlier (*occupational history*) may be the vital clue. If his complaints are those of a severe anaemia, the fact that he has been treated with chloramphenicol (*treatment history*) may be all-important. If he has a fever, the fact that his plane put down in West Africa (*social history*) may be the clue. These are rare examples; more commonly it may be his *social circumstances*—his relations with his wife or his employer—that are at fault. When students start case taking they are wise to make at least some enquiry under all the headings listed. When they have had more experience, they may know on which they should concentrate; but in a difficult case even the most experienced doctor would be unwise to neglect any of the headings listed.

In a simple case—provided one can be sure that the case in point is simple—a few direct questions may obtain all the necessary information. In a difficult one it is best just to let the patient talk, even if the process seems time-consuming. It has been said that one should 'listen to the patient telling one the diagnosis'. Many doctors have had the experience of calling in an eminent consultant, from whom they expected some ultra-modern expertise, only to have the great man sit down, take a good history and solve the problem.

History taking is still an art and a *special form of the art of communication*. It is necessarily a two-way business. At an intellectual level it is important that doctors and students, as well as patients, should confine themselves to words the meaning of which cannot easily be misunderstood. At a deeper level there is no doubt that some doctors inspire confidence in patients and some do not. A better understanding of what is referred to as the doctor-patient relationship can be developed by active participation in sensitivity groups of the kind pioneered for doctors in this country by the late Dr Michael Balint. Members of such groups hope to gain insight into their own personalities and so to acquire greater skill in interpersonal relationships in general and in the doctor-patient relationship in particular.

Doctors often tend to overlook the importance of *non-verbal forms of communication*, which play a big part in their relationships with their

patients. The eyes sometimes convey more information than words; the clenched fist may demonstrate latent tension, and touch may be equally important.

There is a taboo in our culture against the touching of other humans, though this does not extend to horses and dogs. If you are one of those with inhibitions about touch, then on the next occasion that you visit a frightened old lady in hospital try holding her hand. You may well find that when you want to withdraw it, she will grip so that release is difficult; and next time you visit, the hand will be there waiting to be held. For the old lady finds the touch of your hand much more comforting than all your carefully phrased verbal reassurance.

When it comes to physical examination, does the patient feel the doctor's touch as an attack or a caress? There is nothing new in such a suggestion: a distinguished surgeon wrote that abdominal palpation, to be successful, must be like a caress. Patients often derive great satisfaction from being examined, albeit just the taking of the pulse or blood pressure. It may well be that non-verbal means of communication of this sort go a long way to explaining why some doctors inspire more confidence than others.

It is important to realize that *apparent evasiveness* on the part of the patient is almost never deliberate. It can occasionally be due to sub-normal intelligence, but is much more often due to nervousness or actual fright. One must realize that a visit to the doctor or by a doctor is a real ordeal for some patients, quite as bad as a *viva voce* for some students; for the doctor, like the examiner, may be felt at the margins of consciousness to be an all-powerful figure. Thus on approaching a patient who does not know you, tell him who you are and why you have come to see him. Greet him by his own name whenever possible. It is often wise to open the interview with some entirely non-committal chat in the hope that a friendly relationship and some confidence may be established. The discovery of a common county of origin or a common interest in bowls may work wonders.

There is another and more subtle reason for apparent evasiveness. Sometimes the symptoms are psychological ones and the patient has difficulty in describing them precisely. Moreover, physical illness is respectable but many people and some doctors consider that psychological symptoms are not. There is therefore a tendency to present the doctor with a physical symptom, though the complaints are really psychological, and the patient comes to the doctor with a rather vague physical discomfort. Only later may he be able to say that everything is flat and hopeless, and that life is not worth living, or in other words that he is depressed. An anxious patient may present with discomforts in any system or organ in the body, but only later may he be able to voice his ever-present feeling that something awful is going to happen.

Indeed this feeling has become so much a part of him that he is almost unaware of it. Evasiveness is, of course, particularly common when the real problem is a sexual one. It is important that the doctor recognizes the reason for the apparent evasiveness of his patients and does not get angry with them. Unfortunately doctors vary greatly in their ability to tolerate patients with psychological problems. If a patient does in fact make one angry, it is useless simply to try and suppress the feeling. It is much better to say 'I am afraid I find what you are saying difficult to follow. Let's start again.'

In spite of a huge proliferation of ancillary aids, *history taking and physical examination remain essential skills for the doctor*, if only for two simple reasons. Those who work in large modern hospitals are apt to forget that most of the world's population have to be treated either where there are no ancillary aids or where these are very restricted. Even where these aids are freely available, they are in varying degrees costly and are not without discomfort or risk to the patient. It was the original author of this book who wrote: 'From making the cure of the disease more grievous than the endurance thereof, good Lord deliver us.'

Special investigations should therefore be used with discretion; that is, they should be used selectively, when simpler clinical methods have indicated what further investigations are likely to be profitable. It is our view too that no doctor should request any special investigation unless he knows what information relevant to the problem it is likely to provide, and has some idea of its cost and of its possible dangers to the patient.

Thus case taking—the taking of histories and the performance of physical examinations under supervision during the period of medical clerking—remains an essential part of medical education. In this first chapter we deal with history taking and physical examination in general terms. In later chapters we shall elaborate by considering different systems individually and the book ends with a short section about cooperation with the laboratory.

There is no one method of history taking applicable to all patients in all situations. The method will vary according to whether the history is being taken in the wards, the out-patient clinic, the surgery or the home, and according to the state of the patient and the time available. It is wise to open a non-urgent consultation with some general question such as 'What can I do for you?' This gives the patient an opportunity to say what he wants from the consultation. To know this at the outset may be valuable and may indeed save time. Here we attempt to describe the methods that we think should be used by students undertaking medical clerking in hospital. We shall consider these for convenience under the headings mentioned above, but must emphasize once more than patients

cannot be tied down to an orderly sequence. They must be allowed as far as possible to tell their story in their own words and in their own way. Only when they have done this should they be asked to enlarge on what appear to be the more important aspects of the story and only after that should specific questions be asked. Personal history taking must never become a stereotyped routine of asking standard questions and recording the answers, though questionnaires may be useful for special purposes.

The presenting complaint

Try to define the main complaint and its duration. The presenting complaint is simply the complaint which made the patient come to the doctor. The remarks above about apparent evasiveness may apply here, but most patients with physical disease have no difficulty. They have pain in the belly or headache or shortness of breath. In writing up the history students should avoid the temptation to include a mini-history under the presenting complaint. In most cases there is one symptom which made the patient come to the doctor and ideally this is all that should be included under this heading. One must admit, however, that some patients have more than one main complaint and a few have so many that it is impossible to identify a presenting complaint.

The question of duration may be difficult. First, many people, particularly the elderly, cannot remember the duration of their symptoms. One could indeed regard this as the normal state of affairs and suspect the patient who can remember every detail of his illness of being unduly introspective and hypochondriacal.

Most patients with long-standing symptoms tend to date them by events rather than by years, even though there is no causal relationship. The symptoms started 'after my husband died' or 'at the time of the first moon landing'. With patience it is possible to get at the likely duration. After that it is wise to ask some such question as 'Did you ever have anything wrong before that?' or even 'When were you last perfectly well?' In this way earlier symptoms, which the patient regards as unimportant, may be revealed. One should remember too that patients often use the word 'chronic' to mean 'severe', rather than of prolonged duration.

The history of the present illness

Ask the patient to tell you the story of his illness from the beginning. Ideally you should allow him to do this without interruption, but this may be a counsel of perfection. It may require some tactful encouragement to make a dour or nervous patient tell you his story at all, while some talkative ones cannot keep anywhere near the point. A particular difficulty is the patient who will use pseudo-medical terms or terms of

which he does not know the medical meaning. Patients who insist on talking about rheumatism, migraine, acidity, catarrh or disc should be gently discouraged and asked to describe what they actually feel to be wrong. Others will insist on talking about 'what my other doctors said'.

When the patient has told you all that he will spontaneously, ask him to enlarge on any points that you may think to be important. Also try to clear up any doubt about the time of onset and the duration of the main symptoms. Some patients have symptoms which come and go, and it is important to try to find out whether the relapses and remissions are related in any way to times, seasons or events in the patient's life.

When you think that you have the patient's story clearly, you should take each main symptom in turn and examine it in detail. The first step is to try to make sure that you and the patient are talking about the same thing. A patient may, for instance, complain of wind or flatulence. Since flatus is considered in our culture to be an indelicate subject, it may be assumed that he means bringing up wind, whereas he may well mean passing wind by the bowel; or he may mean that he has a feeling that he wants to get rid of wind but is unable to do so. One must therefore enquire directly: 'Do you mean that you bring the wind up or that you pass it down or that you feel that you want to get rid of it but can't?' It should be unnecessary to point out that one should avoid leading questions, which themselves suggest the answer expected, as far as possible, but some questions, such as 'Have you ever spat blood?' may be essential.

Perhaps the commonest complaint which brings a patient to a doctor is a *pain* of some sort and this will serve to illustrate one way in which this nature of a symptom can be further explored.

Ask about the following points:

Site. Where is it? Note whether the patient points to one spot or spreads his hands over a wide area.

Radiation. Does it stay in one place or does it move or spread?

Severity. Does it interfere with his activities and does it ever keep him awake at night? If it never interferes in any way with his activities and never keeps him awake at night, he is probably talking about what should properly be called discomfort. Patients who use such exaggerated terms as continual agony are usually seeking sympathy and one should try to discover the real reason for their distress, which is often social or psychological.

Timing. When did it start? When does it come and when does it go?

Character. What is it like? Most descriptions of the character of a pain, e.g. stabbing, burning, pricking or gnawing, are unhelpful. A distinguished professor of medicine pointed out that to describe a pain as gnawing really conveys no information unless the listener has had the experience of being gnawed. Yet, said the professor, some patients will not only describe a pain as gnawing but will 'specify the species of rodent concerned'. On the other hand the distinction between a colic, which waxes and wanes and may cause a patient to roll about, and a steady pain like that of peritonitis, which causes the patient to try to avoid all movement, may be very important.

The following points are often particularly helpful in deducing what disturbance of normal function is responsible for the pain:

Occurrence or aggravation. What brings it on? And what makes it worse? A pain in the centre of the chest which always comes on after a certain amount of exertion, or is made worse by exertion, is almost certainly due to ischaemia of the heart. A very similar pain which comes on a short time after eating is probably oesophageal.

Relief. What makes it better? Pains may be relieved by simple measures. Pain arising in the musculoskeletal system for instance, may be relieved by a change of position. Upper gastrointestinal pains, e.g. duodenal ulcer, are usually promptly relieved by eating. Lower gastrointestinal pains are relieved by defaecation or the passage of wind. Many cardiac pains, brought on by exertion, are relieved by rest. Any definite relief by simple things of this sort may be a valuable clue to the disturbance of function or structure involved.

The effect of drugs may also be of diagnostic value. Ischaemic cardiac pain is usually promptly relieved by trinitrates. Musculoskeletal pains are usually relieved by simple analgesics like aspirin, while discomforts associated with stress and tension are not. Most physical back pains are therefore relieved to some extent by aspirin, while tension pains usually are not so relieved. In the rare event of a back pain made worse by aspirin, one would have to think of an abdominal cause, e.g. a peptic ulcer adherent to the posterior abdominal wall.

Pain is a symptom which can usually only be further explored in clinical terms. It may, however, be possible to explore other symptoms, thirst for example, in more precise physiological terms.

Thirst must first be distinguished from the dry mouth of oral infections or of defective salivary secretion: and occasional patients with neurosis or compulsive water drinking may complain of thirst. But it is most commonly the prime symptom of loss of body water (with or without loss of salt). The principal causes of loss of body water are diminished intake, vomiting, diarrhoea, increased sweating, increased

output of urine and severe haemorrhage. Observation or simple questions will therefore uncover the immediate disorder of function or functions that are responsible for the symptom, but further questions and investigations may be necessary to explain it in terms of pathology.

If, for instance, the thirst appears to be due to loss of body water due to increased urinary output, one must recall that the huge output of urine of low specific gravity of diabetes insipidus and the similar output of urine of high specific gravity (due to glucose) of diabetes mellitus can both cause severe thirst. The passage of large amounts of urine with a specific gravity of 1010 (isotonic with plasma) in renal failure may be sufficient to cause thirst. Hypercalcaemia, by diminishing the action of antidiuretic hormone and so increasing water loss, may produce thirst and finally the administration of diuretics (or even excessive tea or coffee drinking) may promote salt and water loss with increased urine volume and so thirst.

These two examples—pain and thirst—will serve to illustrate different ways in which all important symptoms should be explored, with the object of identifying, if possible, the disturbance of function and/or structure responsible for them.

While concerning oneself with the details of a patients' symptoms it is important not to lose sight of what may be called the shape of the illness. Is it something that began insidiously and has gradually got worse up to the present time? Or something intermittent? Or something which began acutely and is slowly getting better, but has not yet gone? Sometimes this shape of the illness may be more significant than individual symptoms.

Another valuable question in patients with long-standing symptoms (particularly psychological ones) is: '*What made you decide to come and see me at this particular time?*' The answer is sometimes illuminating. It may well be: 'I wouldn't have dreamt of coming, but my wife made me', or it may be because an acquaintance has recently died with what appeared to the patient to be rather similar symptoms.

On pp. 12–17 there is a scheme, arranged under systems, of the kind of questions that doctors usually ask when taking a history. Such a scheme is of limited value. When the patient has told his story and the stage of examining individual symptoms is reached, the doctor should be asking himself first 'Are we both talking about the same thing?' and then, 'What does this thing (i.e. symptom) mean in terms of disturbance of function and/or structure?' and should be framing his questions accordingly, rather than be repeating a list of standard questions. It is usual, however, to conclude the history of the present illness with a brief review of the other systems which do not appear to be implicated.

Previous history of illness

This should include all important illnesses from infancy onwards. Beware of accepting ready-made diagnoses, particularly as explained above, in the case of such terms as influenza, arthritis, rheumatism and so on. Even if more precise diagnostic terms are used, it is wise to ask a few questions about the nature of the illness to check whether the diagnosis seems likely. Questions about common infections should usually include a tactful enquiry about venereal disease and its treatment.

In some cases it may be necessary to communicate with doctors or hospitals that have treated the patient in the past, either for research purposes or to obtain information necessary for correct treatment. The name and, if possible, the address of the doctor or hospital concerned with the treatment of a previous illness should therefore be recorded, together with the name and address of the patient at the time of the previous illness, if this has changed in the interval, and in the case of a married woman her name before marriage.

The menstrual history

Women should be asked about menstruation. In the majority of cases menstruation occurs every 28 days, but the intervals may be longer or shorter according to the patient's habit. Ask at what age menstruation began and if menstruation has ceased, ask how long it has been absent. The menopause usually occurs about the forty-fifth year or later. Enquire also whether the patient is losing more or less blood than usual. The menstrual flow is to be regarded as abnormal if it lasts for fewer than 2 or more than 8 days. Ask about premenstrual tension and about the presence or absence of pain at the periods; and ask whether the patient has been taking oral contraceptives.

The treatment history

This should include details of drugs taken, including psychotropic drugs, surgery, radiotherapy and psychotherapy. Adverse reactions to drugs, including hypersensitivities (especially to penicillin) are most important. A major difficulty is that patients may not be able to remember, nor may any record exist, about past treatments. Many of his remedies may in any case have been bought across the counter. In this case, and probably always, it helps to ask about remedies taken for particular complaints, e.g. 'What do you take when you have a headache?'; 'What do you take for the bowels?' Patients' memories can be aided by asking them to bring up the whole contents of their medicine store. It is often extensive and outdated. Relatives often remember more than the patient if he is very old, very young or mentally sick. It is important to discover not only whether someone was given a drug to

take, but whether he took it and for the appropriate time. If the drugs were not taken as prescribed, the reasons for them not being taken must be discovered. Patients frequently do not comply with instructions. Vocabulary matters: medicines often mean something liquid or something sold in a pharmacy; drugs may mean doctors' prescriptions or illicit narcotics to a patient; and tablets may not be distinguished from capsules. It is often better to ask a patient what remedies he took for particular complaints and whether these ever disagreed with him, than to use technical terms. Drug manufacturers can usually help with the names of unusual drugs imported from overseas. Much difficulty would be avoided if all doctors used official rather than proprietary names.

The family history

Note the patient's position in the family and the ages of the children if any. Usually it is only necessary to record the state of health, the important illnesses and the cause of death of immediate relatives. If, however, there is any question of an hereditary disorder one should enquire about all known relations and attempt to construct a family tree showing those affected and those not affected.

The social and occupational history

Enquire about what may be grouped together as the patient's physical and emotional environment, his surroundings both at home and work, his habits and his own mental attitude to life and to his work. Try to visualize his life, sharing his emotions and viewing step by step his home, family, daily habits, diet and work. It may help to ask him to give an account of a typical day. As about:

1. The *exact nature of his occupation* (not just the name of his trade but what precisely his work involves) and whether it exposes him to injurious influences. Former occupations should also be noted. One should ask about his attitude to his work, his employers and his work-mates. Sometimes one should enquire into a patient's business affairs and the possibility of financial worries.

2. His *domestic and marital relationships*, his feelings about other members of his family, his interests, hobbies, hopes, fears, the holidays he gets and whether he enjoys them, the amount of exercise he takes, the games he plays, and, in general, the sort of life he leads and the sort of person he is.

3. His *home surroundings*, their sanitary condition and the possible existence of overcrowding or of loneliness. What pets does he keep? Where did they come from and were they recently imported?

4. His *diet* and his *use of alcohol and tobacco*. It is important to ask about past habits in these respects. A man who says that he neither

drinks nor smokes may have been a heavy drinker or smoker in the past. Remember too that many alcoholics will convincingly deny their dependence. Distinguish between cigarette smoking and pipe or cigar smoking.

5. *Whether or not he has lived abroad* and if so whether he was ill there. Recent travel may be important; a patient may for instance suffer from malaria in the UK if he has recently travelled from or even through a malarious area.

The psychiatric history

Patients coming to a general physician may resent psychiatric case taking. It is important therefore to introduce it in an acceptable form. Most patients find it reasonable if the doctor says that pains (or headaches, breathlessness, dizziness—whatever has been complained of) may sometimes be made worse by worry: 'Have you any special worries?' If 'yes' follow up the reply with appropriate supplementary questions. If 'no' ask tactfully worded leading questions about *work, social and sexual relationships*. These are the three main areas of human concern. Other common psychoneurotic symptoms may be asked about directly such as anxiety, specific fears (phobias), obsessive thoughts and compulsive acts and depression. A useful leading question if severe mental disturbance is suspected is: 'Have you had any unusual experiences recently?' which may stimulate the patient to talk about false beliefs (delusions) or false sensations or perceptions (hallucinations). A question about getting on with people may disclose paranoid ideas (of persecution). Much of a psychiatric history is covered by the headings used in taking a medical history: complaint, history of present illness, previous history of illness, family history, etc. The main exception is the personal history. More detail is gathered about early family life, schools, further education, jobs, marriage and children. In the case particularly of children and old persons, information from relatives or neighbours may be important.

There are two more points about history taking which should be mentioned. *First* it is sometimes as important to record that a symptom was not present as to record that it was present. Under each system therefore the absence of the most important symptoms, e.g. breathlessness and cough in the case of the respiratory system, breathlessness on exertion or cardiac pain in the case of the cardiovascular system, and paralysis, headache or fits in the case of the nervous system, should be recorded.

Secondly the history does not end when the patient is first seen. Continuation notes should record the disappearance of symptoms or the

appearance of new ones, or any other relevant fact which becomes apparent while the patient is under observation.

There follows a scheme of the kind of questions which most doctors ask in taking a history.

ROUTINE QUESTIONS

Gastrointestinal system, abdomen and pelvis

1. Symptoms point to an affection of the *upper alimentary tract*. Inquire about:

Pain. What is its severity and site? Is it localized or diffuse? Does it radiate in any particular direction? For how long has he had it? Does he have intervals of freedom? If so, for how long? What is its relation to meals (if any)? Does it wake him at night? What things aggravate it? What affords relief (e.g. food, antacid powders, vomiting)? Distinguish especially between 'pain' and 'sense of discomfort' or 'fullness'.

Appetite. Is it increased or reduced? If reduced, is his appetite really bad, or is he afraid to eat on account of pain?

Weight. Is the weight increasing, decreasing or remaining stationary?

Vomiting. Frequency. Its relation to pain; does it relieve pain or not? Distinguish between vomiting (contraction of abdominal muscles and diaphragm) and regurgitation (contraction of stomach muscles against closed pylorus).

General characteristics of vomited matter. Its amount and colour. Does it ever contain blood? Does it ever look like 'coffee-grounds'? Is it ever sour and frothy? Does it contain residues of food taken the day before?

Flatulence. Does the wind tend to escape downwards or upwards? Does either form relieve the symptoms?

Water brash. Does he ever experience excessive secretion of saliva into the mouth, with regurgitation of mouthfuls of clear, tasteless fluid?

Heartburn. Does he suffer from pain behind the sternum? Does it come on especially when he is lying down?

Dysphagia. Is there any difficulty in swallowing? If so, where does the food appear to 'stick'? Is it worse with liquids or with solids?

2. Symptoms point to an affection of the *lower alimentary tract*. Inquire about:

Diarrhoea. Number and time of occurrence of motions during the day; their relation to meals or to special articles of food. Colour of the motions; are they

formed, unformed, porridge-like, frothy, or frankly watery? Do they float in the lavatory pan or are they difficult to flush away? Has he ever passed any blood or slime? Is there pain during defaecation? Does the patient use purgatives or does he take anything else, e.g. beer, likely to produce loose motions?

Constipation. What is his usual bowel habit? Has there been any recent change in habit? If so, can this be explained by change in diet, medicines, etc.? Does the constipation alternate with diarrhoea? If so, can this be explained by the taking of purgatives? Has he any colicky pain? Has he passed blood? Has he had any vomiting? Does he take codeine in any form?

Pain. Site, radiation and character? Persistent or intermittent? Where is it felt worst? Is it relieved by defaecation or by the passage of flatus?

3. Symptoms point to an affection of the *liver* or *gall bladder*, e.g. patient is jaundiced or has pain in the region of the liver. Inquire about:

Jaundice. Has he noticed any change in the colour of the urine or faeces? Does the skin itch? Have there been any other cases of jaundice among his family, friends or workmates? Has he had any kind of injection in the last 3 or 4 months?

Pain. Its site. Has he ever had any attacks of very severe pain, coming on suddenly and lasting for a few hours? If so did the pain radiate and in what direction? Was he yellow after it subsided? Has he ever had pain in the tip of the shoulder.

Inquire also regarding his digestion on the lines already laid down.

4. Symptoms point to an affection of the *genital system*:

Patients will usually talk spontaneously about structural things, such as a urethral discharge, swelling of the testicles or ulceration of the penis or scrotum in the male; and gynaecological abnormalities in the case of the female. (See specialized textbooks.)

Often, however, their real complaint is of a disorder of function which is to them intensively personal and embarrassing. Tact and sympathetic listening may enable such patients to discuss disorders of coital function, such as impotence, premature ejaculation or frigidity, problems of infertility and possible anxieties about masturbation or homosexuality.

Cardiovascular system

If the symptoms point to an affection of the *circulatory system* inquire about:

A history of *rheumatic fever* or *chorea*. (If a child, ask also about sore throats and 'growing pains'.)

The following subjective sensations:

Dyspnoea. When does it come on? Is it present at rest or only on exertion? What degree of exertion is necessary to produce it? Does he have attacks of breathlessness at night? Does he had to sit up in bed, or can he sleep lying down? Pain or distress; its exact site and character; does it radiate or not? If so, in what direction? What precipitates it, and what, if anything, relieves it?

Palpitation. What brings it on, and how long does it last? Does the heart give an occasional thump now and then? Has the patient ever felt his own pulse during an attack? Enquire also about cough, sputum and haemoptysis, as under respiratory system.

Do the feet swell?

Does he ever have pain in the calves? Or is there undue coldness, redness or blueness of the extremities?

The blood

If the symptoms and appearances point to an affection of the blood, inquire about:

Family history of bleeders. Has he had any loss of blood? Has he been taking aspirin in any form? Are the stools ever black? Has he bleeding piles? (If a woman, is menstruation excessive or diminished?) What kind of a diet does he eat? What drugs has he been taking and to what chemical substances is he exposed in his work or home?

Such subjective sensations as breathlessness on exertion, headache, giddiness or palpitation.

Do the feet swell?

The respiratory system

If the symptoms point to an affection of the *respiratory system*, ask about:

Family history of tuberculosis or allergies; occupation (including past occupations) and possible exposure to animal, mineral or vegetable dusts. Ask particularly about smoking habits, past and present.

Cough. Whether dry or productive. Worst at which time of day? Worsened by any particular conditions, such as cold, dust or pollen? Painful or not?

Sputum. Quantity. Most produced at what time of day? Consistency, colour, and odour. Purulent or not? Ever blood-stained, and if so whether with streaks or clots, and on how many occasions?

Dyspnoea. Under what conditions is it present? At rest, or after varying degrees of exertion? Various 'grades' of dyspnoea have been described, but for the student it is best to inquire from the patient what sort of activity (e.g. walking upstairs, running for a bus, etc.) produces dyspnoea. Patients with severe pulmonary disease may be dyspnoeic at rest. They may also be dyspnoeic at night.

Wheeze. Wheezing may be associated with dyspnoea. The student should inquire when wheeze occurs. Is it constant or intermittent? Does anything provoke it? Is it worse at any particular time or day or night?

Chest pain. Where is it? Is it aggravated by deep breathing or coughing? Was it associated with increase in cough, sputum or dyspnoea? Was the onset sudden, as in spontaneous pneumothorax?

The urinary system

If the symptoms point to an affection of the *kidneys*, e.g. oedema, or *urinary system*, e.g. pain on micturation, ask about:

History of tonsillitis or previous renal disease. Family history of renal disease or high blood pressure. What analgesics has been taken?

Has he any pain in the lumbar region or any attacks of acute pain shooting down into the groin or testicles?

The following remote symptoms: headache, vomiting, drowsiness, paralysis or fits, dimness of vision, dyspnoea.

Does the face ever look puffy in the morning? Are the ankles swollen?

What is the state of the bowels?

Inquire regarding micturition, as follows:

Urine. Is it altered in amount? Does he have to get up at night to pass it?

Is it altered in colour? Is it clear or turbid when passed? Ever any blood in it? If so, at what period of micturition is it present? Is it frothy?

Is there any increased frequency of micturition? Is the increase by day or by night? Is there an increase in volume passed? Is frequency associated with undue thirst? Distinguish between *polyuria* as in diabetes and chronic renal failure and *frequency* as in cystitis.

Is there any pain during micturition? Is it before, during or after the act? What is its character, and where is it felt?

Skin diseases

Inquire carefully into the patient's personal habits as regards diet, clothing and washing. What is his occupation? Does he handle chemical substances or other irritants? Ask if he has been taking any drugs recently. It may be necessary to inquire carefully regarding syphilis. Does the eruption itch? If so, when is the itching worst? Did the eruption appear all at once or in crops? Does he suffer from asthma, hay fever or any other allergic conditions? What are his hobbies? Is he in contact with animals, with insects, with plants? What has he applied to his skin on his own initiative, or on the instructions of his doctor? What cosmetics does she use? Is there a *family* history of skin disease, of asthma, of hay fever, of urticaria? Is there a family history of loss of hair or of excessive hair?

The nervous system

If symptoms point to an affection of the nervous system, ask about:

A family history of mental illness, paralysis, or fits. The nature of the patient's work; is he exposed to any poisons, e.g. lead, mercury, manganese, carbon disulphide or other volatile substances? Syphilis and alcohol should be inquired about. Has he been exposed to tropical infestations?

In cerebral cases it is important to inquire regarding discharge from the ear, and the possibility of a head injury.

Should he complain of *fits*, *attacks*, or *blackouts* the following questions should be asked in order to clarify the nature of the attacks:

Age at first attack? Describe the first attack. When did the second occur? What has been shortest and longest interval between attacks? Do they occur in sleep or not? Has he any premonition or aura? What is its character? Does the patient go rigid? Does he lose consciousness? Is the onset sudden or gradual? Are convulsions present? Are they general or local? Where do they begin and end? Does he fall? Has he ever hurt himself? Does he bite his tongue, micturate or defaecate during the fit? Are there any after-symptoms, such as sleep, headache, automatism or paralysis? Is there any subsequent mental disturbance? Because these patients are seldom clear as to the exact nature of their fits, it is essential to interview separately a reliable person who has seen the patient in a fit. The word 'fit' is undesirable at a first questioning and 'attack' or 'seizure' is preferable.

If he complains of *paralysis*, inquire regarding:

Symptoms of heart disease, hypertension, or diabetes (see Circulatory and Urinary Systems). Had he any premonitory symptoms before the onset? How did the paralysis come on? Suddenly or gradually? Has he any headache or vomiting? Where is the headache situated? (Other subjective symptoms of nervous disease are considered in Chapter 9.)

The locomotor system

If the symptoms point to an affection of the bones or joints:

Inquire for previous manifestations of rheumatoid arthritis, rheumatic fever or gout. Ask about possible associated conditions in the skin (e.g. dermatomyositis, psoriasis, disseminated lupus erythematosus, scleroderma and erythema nodosum), the bowels (ulcerative colitis) and the eyes (e.g. conjunctivitis, uveitis and Sjögren's disease). Ask about the presence of a urethral discharge in the male or of leucorrhoea in the female. Has the patient been exposed to rubella? Tuberculosis and syphilis may occasionally be important. Is there a family history of gout or other rheumatic disorder?

If there is pain referred to a bone, ask whether it is worse in the day or in the night. Bone pains are often described as being deep and boring.

If the pain is in a joint, ask whether it is present constantly or only when the joint is moved. Has the joint been visibly swollen? Does the pain move from one joint to another, as is characteristic of acute or subacute rheumatism?

Children

If the patient is a young child, the following special questions should be put to the mother or other responsible person:

How many other children are there in the family? What are their sexes and ages? Have there been any miscarriages or stillbirths? If so, when? Is there a history of illness in the parents or siblings, or in the parents' near relatives?

Was the mother well during pregnancy and did she take any drugs? Was this a full-term infant? What was its birth weight? Was it born at home or in hospital, and was the labour normal? Were there any unusual symptoms, such as jaundice, cyanosis or fits, in the newborn period?

Was the baby breast-fed, and for how long? If bottle-fed, what type of milk was used? Were vitamin supplements given? When was mixed feeding introduced? Was there a satisfactory weight-gain in infancy? What immunizations were given, and when?

It is particularly important to inquire about the 'milestones of development' (p. 321). When did the baby first smile, sit up, walk and talk? When did he acquire control of bowels and bladder?

What are the child's present habits with regard to eating, sleeping, bowels and micturition? What is his general behaviour like in comparison with his siblings or other children of the same age? If the child is of school age, does he attend school regularly, does he get on well with his lessons, and does he like school?

Has the child ever been separated from his mother? If so, when and for how long? What is the social background? Are the living conditions satisfactory? Does the mother go out to work? If she does, who looks after the child while she is away? Is this an immigrant family? If so, where do they come from, and how long have they been in this country?

Finally, inquire about previous illnesses, their nature and severity, and the ages at which they occurred—infectious diseases, fits, bowel disturbances, upper respiratory infections, discharging ears. If there is a history of cough, was it spasmodic, associated with vomiting, particularly bad at night? And was there a whoop? What drugs has the child received? Has the child ever been in hospital? Have there ever been any accidents involving physical injury, burns or poisoning?

In taking the history try to discover what is really worrying the parents. It may be something deeper than is suggested by the child's symptoms, for example leukaemia or a serious disease which affected another child in the family.

The occupational history

The following notes on taking of an occupational history will be found helpful.

In clinical practice the occupational history is often valuable, and there are few surer and quicker means of gaining a patient's confidence than the display of an intelligent knowledge of his job. It is a wise rule to take the occupational history from the time the patient left school. Record the dates and items of all subsequent jobs. He may be exposed to a noxious substance responsible for his ill-health in his present occupation, but this should not be assumed. A man describing himself as an ice-cream vendor may have cancer of the skin of the hand due to

work in the pitch-beds of a gasworks 20 years before, or a mesothelioma due to exposure to asbestos many years before. Cancer of the genito-urinary tract may be the result of exposure in the past to certain aromatic amines used as intermediates in the dye-stuffs industry, as an anti-oxidant in rubber and cable-making and in the laboratory handling of such substances as benzidine, once used for occult blood tests, but no longer available. Ask the patient the name of his trade, the processes employed, the tools used and the substances handled. The name of an occupation may be misleading, for different names are used for the same process in different places.

It often happens that workmen and foreman refer to chemical substances by their popular names and not by their chemical names. Examples of such names are lunar caustic for silver nitrate, chrome yellow for lead chromate, wood spirit for methyl alcohol, and oil of mirbane for nitrobenzene. The man may be ignorant of the nature of a substance he uses and know it only by a trade name. In such cases it is best to communicate with his works manager and ask what is the nature of the substance in question.

Question him as to the general conditions at his place of work. If necessary ask him to sketch on paper a plan of his workshop and of the apparatus he uses. Is the job dusty, and if so what tools make the dust? Are there fumes or vapours, and if so what are the chemical substances involved? Most of the toxic substances encountered in the dangerous trades enter the body by inhalation. Ask whether a hood is installed over his bench, and whether it is connected to a suction system. Ask about the provision of protective clothing at his place of work. Does he wear a special suit, gloves or goggles, and why? Finally ask whether any similar illness has befallen a fellow workman.

Whenever serious doubts and difficulties arise, it is advisable to visit the factory in order to ascertain the conditions of work on the spot. In difficult cases the practitioner should enlist the help and advice of HM Factory Inspectorate, through the Chief Medical Adviser, Baynards House, Chepstow Place, London W2 (01-229 3456).

Other aspects of the history are no less important. A particular illness may render a man temporarily or permanently unfit to do his work. The doctor should know that conditions peculiar to certain trades may cause disease which predisposes to infection. Thus, silicosis leads to an excessive mortality from pulmonary tuberculosis and also from pneumonia. Diseases other than infections may be involved. For example, a heavy mortality from cirrhosis of the liver as well as from tuberculosis exists among publicans, barmen, brewers' draymen and others who have ready access to alcohol.

The doctor should have regard for his patient's work, even when he is suffering from a disease which is non-occupational; one must know

whether a man does a job which makes him a danger to others. A dairyman with open tuberculosis can contaminate milk with tubercle bacilli by coughing into it, and those who handle food can initiate outbreaks of typhoid fever, dysentery and *Salmonella* infection by acting as carriers.

THE PHYSICAL EXAMINATION

The examination of the general state of the patient and of the different systems is described in Chapters 2 to 11. Though the findings should be recorded under systems, patients in medical wards are usually examined from above downwards, by the methods of inspection, palpation, percussion and auscultation, as may be appropriate to the different parts of the body. One should therefore develop a routine of physical examination which combines speed with thoroughness, but disturbs the patient no more than necessary. With practice a routine examination of the kind outlined here can be performed in 15 minutes or less. It need hardly be said that the examination must be carried out as gently as possible, without tiring or exposing the patient more than necessary. In the case of severely ill patients it may be necessary to postpone a routine examination and to perform only the minimum necessary for a provisional diagnosis and treatment. Ill patients must obviously be treated with special care and consideration.

Different doctors have different routines for examining patients in different circumstances, and for writing up their case notes.

The following is the kind of routine examination which students are expected to carry out on patients in medical wards in hospital. Such a routine may have to be modified according to the needs of the patient (e.g. minimum necessary examination in an acutely ill patient; complete examination of the nervous system in a patient with neurological symptoms; see Chapter 9) or according to the circumstances (e.g. in the doctor's surgery or in the patient's home).

During the taking of the history and the performance of the examination the following should be observed:

General

General appearance of illness (does the patient look healthy, unwell or ill)

Intelligence (see p. 205)

Mental state (see p. 202)

Expression

Build

State of nutrition, obesity, oedema

Skin colour, cyanosis, anaemia, jaundice, pigmentation

Skin eruptions, petechiae, spider naevi

Body hair

Deformities, swellings
Temperature, pulse, respiration rate

Hair

Area of scalp covered
Texture

Eyes

Simple tests of visual acuity
Exophthalmos or enophthalmos
Ptosis
Oedema of the lids
Conjunctivae: anaemia, jaundice or inflammation
Pupils: size, equality, regularity, reaction to light, accommodation
Eye movements, nystagmus, strabismus
Ophthalmoscopic examination of the fundi

Face

Facies
Function of motor part of fifth nerve
Function of seventh nerve

Mouth and pharynx (a torch and tongue depressor should be used)

Breath odours
Lips: colour and eruptions
Tongue: protrusion and appearance
Teeth and gums (if patient has dentures, notice whether they fit and ask whether they are worn for meals or only for adornment)
Buccal mucous membrane: colour and pigmentation
Pharynx
Movement of soft palate
State of tonsils

Neck

Movements
Veins
Lymphatic glands
Thyroid
Carotid pulses

Upper limbs

General examination of arms and hands
Fingernails: clubbing or koilonychia
Pulse rate, rhythm, volume and character
State of arterial wall of radials and brachials
Axillae, lymphatic glands
Blood pressure

Test for power, tone, reflexes, coordination and sensation

Joints

Thorax

Anteriorly and laterally

Type of chest, asymmetry if any, breasts

Rate, depth and character of respiration

Pulsations

Dilated vessels

Position of trachea by palpation

Look for and palpate apex beat

Palpate over precordium for thrills

Palpate respiratory movements

Estimate tactile vocal fremitus

Percuss the lungs

Auscultate the heart sounds

Auscultate the breath sounds

Estimate vocal resonance

Posteriorly (patient sitting)

Inspect and palpate respiratory movement

Estimate tactile vocal fremitus

Percuss the lung resonance

Auscultate the breath sounds

Estimate vocal resonance

Note movements and deformities of the spine

Palpate from behind cervical glands, thyroid

Look for sacral oedema

Abdomen

Inspection: size, distension, symmetry

Abdominal wall: movement, scars, dilated vessels

Visible peristalsis or pulsation

Pubic hair

Hernial orifices

Palpation: tenderness, rigidity, hyperaesthesia, splashing, masses, liver, gall bladder, spleen, kidneys, bladder

Percussion: masses, liver, spleen, bladder

Auscultation: bowel sounds, murmurs

Impulse on coughing

Inguinal glands

Genitalia: penis, scrotum, spermatic cord

Abdominal reflexes

Rectal examination when indicated

Gynaecological examination when indicated

Lower limbs

General examination of legs and feet

Oedema

Varicose veins

Test for power, tone, reflexes (including plantar response), coordination and sensation

Joints

Peripheral pulses

Temperature of feet

Examination of excreta

Urine, sputum, stools, vomit: examine by naked eye and measure or estimate amount

Test urine for specific gravity, sugar, protein and blood

WRITING OUT THE HISTORY AND EXAMINATION

Different doctors do this in different ways. The aim is to write a complete yet concise record of a patient's illness. For easy reference this is best done in note form, with important facts starred or underlined.

The physical examination, although performed from above downwards, should be written out under systems. A short statement of the findings under each system should be included, for the absence of signs, as well as of symptoms, can be as important as their presence. The minimum statement about a patient's cardiovascular system might for example read as follows:

pulse 76 regular

neck veins not distended

BP 130/80

apex beat not displaced

heart sounds I and II heard in all areas

no murmurs

Simple line drawings can often convey more information than much writing.

It is usual for a student to conclude his writing-up of a case with a list of tentative diagnoses and a list of investigations required, followed by continuation notes.

This part of the medical record in particular has recently been much criticized. Those interested are recommended to read 'Can we write better notes? An introduction to problem orientated medical records (the Weed approach).' *British Journal of Hospital Medicine* (1972), 7, 603. But the decision to adopt POMR, as it has come to be called, is really one for the department or unit rather than for the individual student.

PRESENTING A CASE

The value of a student's or doctor's notes on a case is much diminished if he is unable to communicate them in a concise form to other students

or doctors. Students should therefore practise making a short summary of their findings, emphasizing both important positive findings and relevant negative ones. The summary should always begin with the name, age, sex and occupation of the patient, and can with advantage end with a brief statement of the problems to which the findings have given rise.

INTERPRETATION

The object of history taking, physical examination and ancillary investigations is the *making of a diagnosis*. In the past this has often been taken simply to mean the detection of a disease process, e.g. 'This patient has Hodgkin's disease'. If no disease process was detected the complaints were described as functional. This was illogical since all disease involves some disorder of function. Further, though a majority of the highly selected patients in hospital wards may have a detectable disease process, this is by no means true of all the patients who consult a doctor; and even when a patient has a disease process, his disability may be quite out of proportion to the physical changes present—a state of affairs sometimes referred to by the unfortunate term 'functional overlay'. The reasons for such overlay should be precisely stated.

One should begin the process of making a diagnosis by asking oneself some such broad questions as 'What is this person's problem?' and 'Has he a disability?' If one decides that he has a disability, one should ask:

1. *How far* can this person's disability be explained by his environment, i.e. in geographical, socio-economic and cultural terms?
2. *How far* can this person's disability be explained by his own attitude and mental make-up, i.e. in psychological terms?
3. *How far* can this person's disability be explained by a disease process or processes, i.e. in pathological terms?

Such an approach is clearly more reasonable than to search for a disease process and then to label as functional any disability not so accounted for.

It used also to be axiomatic that one should try to account for all a patient's symptoms by one disease process. But a surprising number of patients have in fact more than one (e.g. coronary artery disease and an hiatus hernia, both of which may produce central chest pain) and still more have a disease process which either does not explain their symptoms at all or does not explain all their symptoms (e.g. weakness and tiredness in a patient with mild angina pectoris or mild anaemia).

In making a diagnosis one should try to account for a person's total disability and should not be dismayed if this involves mentioning more than one item. Thus the diagnosis in an old lady with multiple symptoms might well be:

1. Loneliness
2. Depression
3. Mild osteoarthritis

or in a young man with dyspepsia:

1. Impending marriage
2. Anxiety state
3. Duodenal ulcer

A diagnosis of this kind gives a truer picture of the state of affairs than a statement that the patient has osteoarthritis or duodenal ulcer with functional overlay.

The fact that many patients have no detectable disease process or have a disease process which in no way accounts for their disability, does not however diminish the importance of detecting disease processes when they are present. That is the main object of clinical methods and is what the rest of this book is about.

2

GENERAL CONSIDERATIONS AND APPEARANCES

The Physical State — The Mental State

THE PHYSICAL STATE

For a good physical examination one needs reasonable quiet, a warm room and good lighting. It is useless to try to examine a patient who is shivering. Daylight is best for the detection of colour changes; and in particular slight to moderate degrees of jaundice may not be seen in artificial light. For a complete examination the patient should be asked to undress and should then be covered with a blanket or dressing gown. In practice patients are often examined wearing pants or trousers, but this can lead to mistakes. One of the writers completely missed an arteriovenous aneurysm in a young man with a collapsing pulse, and the multiple abscesses of a drug addict in a woman with the nephrotic syndrome, through neglecting to examine the thighs and buttocks. Ideally a chaperone should be present when a doctor is making any examination of a female patient; and this is essential in the case of rectal and vaginal examinations, both to reassure the patient and to protect the doctor from subsequent accusations of impropriety.

When one considers the general appearance, the most important step is to make a rapid assessment of the *degree of 'illness'*. This does not mean to make a diagnosis. One has simply to answer the question: 'Does this patient look well, mildly ill, or severely ill and therefore in need of urgent attention?' Experienced nurses are often highly skilled in this kind of assessment and one neglects their opinions at one's peril. Some severely ill patients complain little; occasionally one meets a patient whose appearance of excellent health belies his protestations of unbearable agony.

It has already been said that a good doctor begins his examination as soon as he meets the patient, and continues taking the history until the consultation ends. The examination of systems may provide information about organs and functions, but in physical examination as well as in history taking it is important to try to view the patient as a whole person.

One should therefore make some assessment of the patient's *intelli-*

gence and of his *mental and emotional state*. Simple tests of intelligence (p. 205) may be necessary and on p. 36 there are notes on the assessment of a person's mental state. Observation, as well as the history, may assist in the assessment of the emotional state. Thus an anxious person may be restless, with wide palpebral fissures and sweating palms. Is the anxiety reasonable in the circumstances or is the patient over-anxious? In rare cases of classical hysteria the patient may have an apparently severe disability, but shows a complete lack of the appropriate anxiety — *la belle indifférence*. The lowered mood, inability to concentrate or make decisions, mental retardation, apathy or even obvious misery of a depressed patient may be clearly evident; but so-called 'masked depression', in which these features are less obvious, is an important cause of physical symptoms.

The *attitude* of a patient may give valuable information. Severely ill patients slip down into the most uncomfortable attitudes and are unable to correct their position for themselves. Patients with congestive heart failure may become dyspnoeic if they lie flat (orthopnoea). Patients with abdominal pain due to peritonitis lie still, while patients with colic are restless or even roll about in futile attempts to find relief. Patients with painful joint diseases often have an attitude of helplessness. Various neurological disorders produce characteristic attitudes (Chapter 9). In the severest cases of meningitis the neck may be bent backwards so that the head appears to bore into the pillow.

The *gait* should be observed in patients able to walk. Important abnormalities of the gait are described in Chapters 9 and 11, but note that simple things like a painful corn, an ill-fitting shoe or a strained muscle may produce a temporary limp.

One should make some assessment of a patient's *build* and *state of nutrition*. Usually it is sufficient to observe whether he is lean or fat and muscular or asthenic. In the indigenous populations of western countries overnutrition is a main problem; but in the rest of the world signs of undernutrition, wasting, apathy, anaemia, rough skin and sometimes signs of specific deficiencies may be seen. These have also been detected in immigrants to the UK and in the elderly.

In medical examinations for insurance purposes it is usual to record the *height*, *weight*, *chest measurement* in inspiration and expiration, and *waist measurement*. If the waist exceeds the chest, either the patient is unduly fat or the abdomen is enlarged by disease, usually by the presence of fluid (ascites). Tables 1 and 2 show 'ideal' weights for different heights and builds, as used for insurance purposes. The general state of nutrition may be assessed with the aid of these tables or by the use of skin-fold calipers or by simple observation.

Observe the patient's *face*. His expression, and particularly his eyes, may indicate his real feelings better than do his words. The character-

istic facies of various diseases, e.g. myxoedema, thyrotoxicosis, third and seventh cranial nerve palsies (pp. 220, 230) and paralysis of the cervical sympathetic (Horner's syndrome) (p. 227), must be learnt in practice.

Parotid swellings are obvious on inspection of the face. The temporary tender bilateral parotid swelling of mumps or the unilateral swelling with reddening of the skin from acute parotitis can be contrasted with the non-tender bilateral persistent enlargement, accompanied by dry tearless eyes, of Sjögren's syndrome, or the more irregular unilateral lump of a mixed parotid tumour.

The *cheeks* give some information regarding the patient's health. In anaemia and aortic regurgitation they are pale; in cases of mitral stenosis there is sometimes a bright circumscribed flush over the malar bones; in many persons who lead an open-air life they are red and high-coloured; in congestive heart failure they may also be high-coloured, but the colour is of a bluish tint which cannot be mistaken for the red cheeks of weather-beaten people. In some cases of disseminated lupus erythematosus there is a red raised eruption on the bridge of the nose extending on to the cheeks in a 'bat's-wing' distribution.

Look at the *skin*. The most important abnormalities in a general examination are pallor, yellowness, pigmentation, cyanosis and cutaneous eruptions.

Pallor depends on the thickness and quality of the skin, and the amount and quality of blood in the capillaries. It is thus seen in persons with thick or opaque skins who are always pale; in states where the blood flow in the capillaries is diminished, such as shock, syncope or left heart failure; and locally in a limb deprived of its blood supply; or in the fingers or toes when arterial spasm occurs on exposure to cold, as in Raynaud's disease. Generalized pallor may also occur in severe anaemia. Anaemia, however, is to be judged 'by the colour of the blood rather than that of the patient' (R. Asher) and the colour of the skin may be most misleading. That of the mucous membranes of the mouth and conjunctivae gives a better indication, and so does the colour of the creases in the palm of the hand. *Yellowness* may be due to haemolytic jaundice, when the tint is pale lemon-yellow, or to obstructive jaundice, when it may be of a dark yellow or orange tint. In obstructive jaundice there may be scratch marks that result from the itching which the bile salts evoke. In rare cases yellowness may be due to carotinaemia. *Pigmentation* is most commonly racial or actinic. Other forms are described in Chapter 8. The classical pigmentation of Addison's disease, which affects the buccal mucous membranes as well as the skin of exposed parts and parts pressed upon, has become very rare. *Cyanosis* is a bluish colour of the skin and mucous membranes due to an increase

TABLE 1. IDEAL WEIGHTS FOR MEN AGED 25 AND OVER

<i>Height</i>		<i>Small frame</i>		<i>Medium frame</i>		<i>Large frame</i>	
ft	in	lb	kg	lb	kg	lb	kg
5	2	112-120	50.8-54.4	118-129	53.5-58.5	126-141	57.2-64.0
5	3	115-123	52.2-55.8	121-133	54.9-60.3	129-144	58.5-65.3
5	4	118-126	53.5-57.2	124-136	56.2-61.7	132-148	59.9-67.1
5	5	121-129	54.9-58.5	127-139	57.6-63.0	135-152	61.2-68.9
5	6	124-133	56.2-60.3	130-143	59.0-64.9	138-156	62.6-70.8
5	7	128-137	58.1-62.1	134-147	60.8-66.7	142-161	64.4-73.0
5	8	132-141	59.9-64.0	138-152	62.6-68.9	147-166	66.7-75.3
5	9	136-145	61.7-65.8	142-156	64.4-70.8	151-170	68.5-77.1
5	10	140-150	63.5-68.0	146-160	66.2-72.6	155-174	70.3-78.9
5	11	144-154	65.3-69.9	150-165	68.0-74.8	159-179	72.1-81.2
6	0	148-158	67.1-71.7	154-170	69.9-77.1	164-184	74.4-83.5
6	1	152-162	68.9-73.5	158-175	71.7-79.4	168-189	76.2-85.7
6	2	156-167	70.8-75.7	162-180	73.5-81.6	173-194	78.5-88.0
6	3	160-171	72.6-77.6	167-185	75.7-83.5	178-199	80.7-90.3
6	4	164-175	74.4-79.4	172-190	78.1-86.2	182-204	82.7-92.5

Heights are measured wearing ordinary shoes and weights in ordinary indoor clothing.
 Notice that tables of this kind make no allowance for 'middle-aged spread'.

TABLE 2. IDEAL WEIGHTS FOR WOMEN AGED 25 AND OVER

Height		Small frame		Medium frame		Large frame	
ft	in	lb	kg	lb	kg	lb	kg
4	10	92-98	41.7-44.5	96-107	43.5-48.5	104-119	47.2-54.0
4	11	94-101	42.6-45.8	98-110	44.5-49.9	106-122	48.1-55.3
5	0	96-104	43.5-47.2	101-113	45.8-51.3	109-125	49.4-56.7
5	1	99-107	44.9-48.5	104-116	47.2-52.6	112-128	50.8-58.1
5	2	102-110	46.3-49.9	107-119	48.5-54.0	115-131	52.2-59.4
5	3	105-113	47.6-51.3	110-122	49.9-55.3	118-134	53.5-60.8
5	4	108-116	49.0-52.6	113-126	51.3-57.2	121-138	54.9-62.6
5	5	111-119	50.3-54.0	116-130	49.0-59.0	125-142	49.4-64.4
5	6	114-123	51.7-55.8	120-135	54.4-61.2	129-146	59.5-66.2
5	7	118-127	53.5-57.6	124-139	56.2-63.0	133-150	60.3-68.0
5	8	122-131	55.3-59.4	128-143	58.1-64.9	137-154	62.1-69.9
5	9	126-135	57.2-61.2	132-147	59.9-66.7	141-158	64.0-71.7
5	10	130-140	59.0-63.5	136-151	61.7-68.5	145-163	65.8-73.9
5	11	134-144	60.8-65.3	140-155	63.5-70.3	149-168	67.6-76.2
6	0	138-148	62.6-67.1	144-159	65.3-72.1	153-173	69.4-78.5

Heights are measured wearing ordinary shoes and weights in ordinary indoor clothing.
Notice that tables of this kind make no allowance for 'middle-aged spread'.

in the amount of reduced haemoglobin in the blood. It may be divided into central and peripheral. Central cyanosis results from imperfect oxygenation of blood, as in heart failure and some lung diseases, or from the mixture of arterial and venous blood in the presence of right-to-left or venous-arterial shunts in the heart. In this case the cyanosis is general and the cyanosed extremities are warm. It characteristically affects the tongue. Peripheral cyanosis is due to excessive reduction of oxyhaemoglobin in the capillaries, when the flow of blood is slowed. This may happen on exposure to cold, when there is venous obstruction or in heart failure. The cyanosed extremity or extremities are then cold and the tongue is unaffected. One should note, however, that the cyanosis of heart failure is often of a mixed type, due to both central and peripheral causes. A similar bluish or leaden colour may in rare cases be produced by methaemoglobinaemia or sulphaemoglobinaemia, usually due to the taking of drugs such as phenacetin. This should be considered in any patient who is cyanosed but not breathless. Carbon monoxide poisoning produces a generalized cherry-red discoloration.

Cutaneous eruptions are described in Chapter 8. Purpuric haemorrhages (petechiae), which do not disappear on pressure, are of great importance in blood diseases and in infective endocarditis. Spider naevi (telangiectases), which have typically a red centre and spidery branching tributaries and which do disappear on pressure, are an important sign of liver disease.

When an excess of fluid is present in the subcutaneous tissue the condition is known as *oedema*. Thus in acute nephritis an early symptom is oedema of the face, which is most marked when the patient rises in the morning. In dependent oedema, however, which is typically present in congestive heart failure, and in conditions associated with a low plasma protein level, the swelling first appears at the ankles and over the dorsum of the foot, and only gradually mounts to the legs, thighs and trunk. In local venous obstruction, the oedema is confined to the parts from which the return of blood is impeded. In this way one finds oedema of an arm when malignant glands constrict the axillary vein or oedema of a leg in thrombosis of the popliteal or femoral vein. Oedema of the whole upper part of the body may result from intrathoracic tumours. Oedema may be recognized by the pallid and glossy appearance of the skin over the swollen part, by its doughy feel, and by the fact that it pits on finger pressure. In recumbent patients oedema often appears first over the sacrum. In eliciting pitting it is important to press firmly and for a sustained period, or slight oedema may be missed.

Localized oedema may be due to local changes in capillary permeability, as in angioneurotic oedema and giant urticaria.

Subcutaneous emphysema is uncommon, but if present can be readily recognized by the crackling sensation which is detected on pinching the part affected.

The *hands* of the patient should be examined (Plate I). Notice the strength of grip as he shakes hands; this often indicates improvement or deterioration with considerable accuracy. Their general shape should be noted, along with the state of the joints, the character of the nails, the presence or absence of finger-clubbing and the presence of staining with nicotine.) In osteoarthritis the finger joints are often implicated, and bony nodules, known as *Heberden's nodes*, are formed at the basis of the terminal phalanges. In rheumatoid arthritis there is characteristically a spindle-shaped swelling of the interphalangeal joints and later an ulnar deviation of the fingers. *Trophic changes* in the skin may be present in neurological diseases and in disorders of the peripheral circulation (e.g. Raynaud's disease). Characteristic movements or attitudes of the hand may also be seen in athetosis, tetany and lead palsy. *Tremor* of the hands may occasionally be congenital. In other cases it is due to nervousness, senility, parkinsonism, thyrotoxicosis, alcoholism, disseminated sclerosis, uraemia, hepatic failure or mercurial poisoning. This and other abnormal movements are considered on p. 252. In ulnar paralysis the hand becomes deformed by over extension of the first phalanges, combined with excessive flexion of the rest, so that a claw-like attitude is produced. This is known as the '*main en griffe*'. Wasting of the small muscles of the hand, due for example to median or ulnar nerve lesions, cervical root (C8) disease or loss of anterior horn cells at the same level, gives the hand a flattened appearance. In *Dupuytren's contracture* there is a thickening of the palmar fascia, which may lead to a flexion contracture of the ring and other fingers. In acromegaly the hands are massive, the fingers being spatulate with square tips, and the skin thickened. In *clubbing of the fingers* the tissues at the base of the nail are thickened, and the angle between the nail base and the adjacent skin of the finger is obliterated. The nail itself loses its longitudinal ridges and becomes convex from above down as well as from side to side. In extreme cases the terminal segment of the finger is bulbous like the end of a drumstick. The condition may occasionally be congenital. Gross degrees of clubbing are found in association with severe chronic cyanosis, as in congenital heart disease; and in association with chronic suppuration within the chest, as in bronchiectasis and empyema. Lesser degrees may be found in carcinoma of the lung, pulmonary tuberculosis, and chronic abdominal conditions such as steatorrhoea, Crohn's disease and ulcerative colitis. Clubbing is also an important sign of subacute bacterial endocarditis, when it may be associated with Osler's nodes, tender transient swellings about the size of a pea in the pulp of the fingers and toes, and 'splinter' haemor-

rhages beneath the nails. In hypertrophic pulmonary osteoarthropathy there is, besides clubbing of the fingers, thickening of the periosteum of radius, ulna, tibia and fibula. This gives rise to swelling above the wrist and ankle. *Koilonychia* occurs in iron-deficiency anaemia. The nails are soft, thin and brittle. The normal convexity is lost and replaced by a concavity.

The *feet* may remain overlooked under bedclothes or socks. Apart from looking for pitting oedema, the condition of the skin of the feet is of importance, especially in diabetics and the elderly. Peripheral vascular disease will make the skin shiny and hair does not grow on ischaemic legs or feet. The dorsalis pedis and posterior tibial pulses may be reduced or absent. If the toes of an ischaemic foot are compressed their dull purple colour will blanch and only slowly return. Painless trophic lesions, often with deep ulceration, on the soles are seen frequently in diabetic peripheral neuropathy.

The *neck* should be inspected and palpated. Swellings in the neck are usually felt best from behind. Note:

1. The state of the *lymphatic and salivary glands*. In infected conditions of the tonsils the glands at the angles of the jaw are enlarged, and those below the jaw in cases of malignant disease in the mouth. Glands draining an inflammatory focus are usually tender. Enlarged tuberculous glands may occur in groups or in long chains beside the sternomastoid, and scars may mark the points of past suppuration in severe untreated cases. In Hodgkin's disease and other reticulososes the glands are enlarged and discrete. In lymphatic leukaemia there may be great enlargement of the glands on both sides. In secondary syphilis the glands under the upper part of the trapezius are often palpable. If enlarged glands are found either in the neck or elsewhere, it is important to observe whether they are firm and distinct, or fused together, whether fluctuation can be elicited, and whether they are adherent to adjacent structures. The *submandibular salivary glands* should also be palpated when the neck is being examined from behind. If they are swollen and tender, the opening of their ducts into the mouth should be inspected with the tip of the patient's tongue rolled upwards; a salivary calculus may be seen.

2. The *thyroid gland*. Inspect the neck for any general or local enlargement of the gland, and observe its movement with the larynx as the patient swallows. Patients find this easier if they are given a glass of water. Then stand behind the patient and palpate the gland with one hand on each side of the neck. Determine if any swelling exists, and if so whether it is uniform or nodular, hard or soft. Sometimes such enlargements press on the trachea and occasionally extend into the thorax behind the sternum; at other times, particularly if the disease is malignant, the recurrent laryngeal nerves may become implicated. In

cases where there is difficulty in determining whether a tumour is connected with the thyroid, it is helpful to remember that the gland and any tumour connected with it moves up and down on swallowing. Minor degrees of enlargement of the thyroid are often better seen than felt. A bruit on auscultation shows that the gland is hyperactive.

3. *Pulsations in the vessels* must be recorded. Any arterial pulsation is both seen and felt as a distinct thrust, whereas venous pulsation is seen but is not felt as a thrust, if it is felt at all. In aortic incompetence the carotid arteries are seen to pulsate forcibly. In aortic stenosis a systolic thrill is felt. Women patients with hypertension sometimes show kinking of the right common carotid artery which simulates aneurysm. The jugular veins may be distended and pulsatile in congestive heart failure (p. 98). In superior mediastinal obstruction due to retrosternal goitre or malignant neoplasm in the mediastinum, non-pulsatile distended veins may be seen over the neck and upper part of the body: cyanosis and oedema of the upper part of the body may accompany this sign. Distended neck veins may also be seen in large pericardial effusions.

The chance of detecting a treatable cancer should make a full *examination of the breasts* a necessary feature of every general examination of a woman. With the patient reclining, arms to the sides, inspect the development and symmetry of breasts and nipples. Look for any reddening of the skin, ulceration or dimpling (*peau d'orange*). Retraction (rather than inversion) of one nipple may signpost the cancer beneath. If there is a discharge from the nipple determine whether it is bloody, serous or milky.

Palpate each breast with the flat of the fingers, working over the whole breast as if it was mapped out in quadrants. Repeat this when the patient has her hands placed behind her head. If a lump is found the qualities to be observed are those that hold good for any lump felt anywhere in the body. Determine the situation, size, shape, surface and edge; feel its consistency and mobility in relation to deep and superficial structures.

Then examine the *axillae*. It is difficult to feel enlarged lymph glands unless the patient's arm is raised to allow the examining fingers to be pushed high into the axilla. The arm is then lowered and palpation is continued downwards along the chest wall. Any swelling of the male breast is likely to be seen at a glance. The swelling can be distinguished as breast tissue rather than pectoral fat by palpation when the patient's hands are behind his head. At some stage of puberty the majority of normal boys will have a palpable disc of breast tissue beneath the areola.

Lastly, it is usual in considering the patient's general condition to observe the temperature, pulse and respiration.

Temperature. When taking the temperature, the following practical points must be attended to:

1. The thermometer must be accurate and of NPL standard.
2. The thermometer must be kept in position long enough to allow the mercury to reach the body temperature. It is advisable to exceed the period which the instrument professes to require. The ordinary 'half-minute' thermometer should be left in position for one or two minutes. Collapsed, comatose and elderly patients should have the rectal temperature taken with a special 'low-reading' thermometer. Accidental hypothermia is not uncommon in the elderly.
3. In conscious adults the temperature is taken in the mouth or in the axilla. In young children the thermometer should be placed in the fold of the groin, and the thigh flexed on the abdomen; or it may be inserted into the rectum. The temperature of the mouth and rectum is generally at least half a degree higher than that of the groin or axilla. When the temperature is taken in the mouth, the patient must breathe through the nose and keep the lips firmly closed during the observation.
4. Before inserting the thermometer, make it an invariable rule to wash it in antiseptic or in cold water, and see that the mercury is well shaken down. Wash it again before replacing it in its case. The Centigrade (Celsius) scale is in general use, although in Great Britain many people are still more familiar with the Fahrenheit scale (for comparison of the two scales see p. 347).

Normal	36·6–37·2°C	(98–99°F)
Subnormal	below 36·6°C	(below 98°F)
Febrile	above 37·2°C	(above 99°F)
Hyperpyrexia	above 41·6°C	(above 107°F)
Hypothermia	below 35°C	(below 95°F)

In many conditions, notably acute fevers, there is a disturbance of heat regulation, which may be looked on as the setting of the 'thermostatic' mechanism controlling heat gain and loss at a higher level than normal. While the temperature is rising to this new level, heat is being conserved, the skin vessels are constricted so that the body surface feels cold, and the patient may even shiver violently. This shivering is referred to as a *rigor*. When the higher temperature is reached, heat loss again becomes apparent; the skin vessels dilate and the body surface feels warm. This is the state of affairs present in sustained fever or *pyrexia*.

There are three classical *types of fever*—the continued, the remittent and the intermittent. When fever does not fluctuate more than about 1°C (or 1·5°F) during the twenty-four hours, but at no time touches the normal, it is described as *continued*. When the daily fluctuations exceed 2°, it is known as *remittent* (Fig. 1); and when fever is present only for

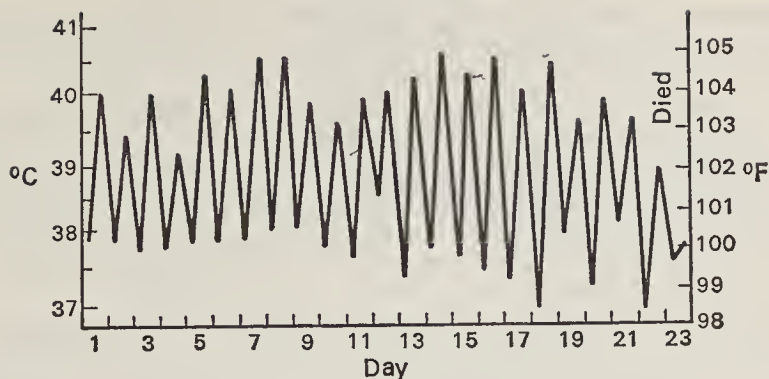


Fig. 1. Remittent fever.

several hours during the day it is called *intermittent*. When a paroxysm of intermittent fever occurs daily, the type is *quotidian*; when on alternate days, *tertian*; when 2 days intervene between consecutive attacks, *quartan* (Fig. 2).

One must add, however, that with the modern use of antibiotics and other specific drugs these classical types of fever are not often seen.

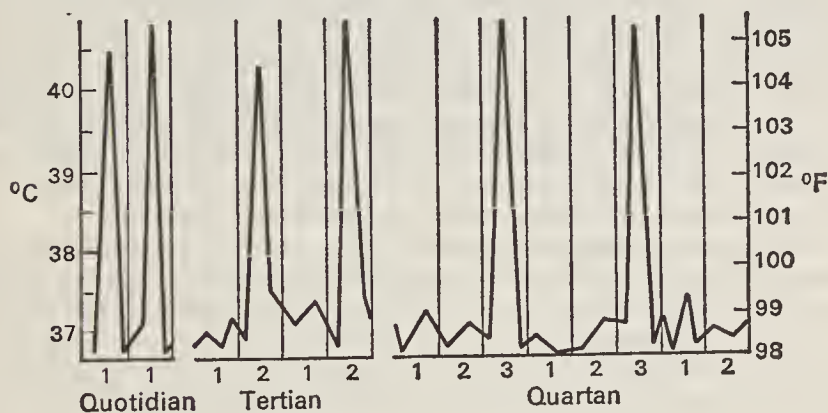


Fig. 2. Intermittent fever.

Pulse. Count it for a full minute when the patient is at rest and composed. Abnormalities due to cardiovascular causes are described in Chapter 5. The rate in health and under conditions of a medical examination varies from about 60 to 80 beats a minute. The common causes of a rapid pulse are recent exercise, excitement or anxiety, shock (e.g. bleeding), fever and thyrotoxicosis. A slow pulse is characteristic of severe myxoedema and of complete heart block.

Respiration. Count the patient's respirations for a full minute when his attention is distracted from his breathing. It is convenient to do this when he thinks that you are still counting his pulse. The normal rate in an adult under the conditions of a medical examination is about 16 to 22 respirations a minute, but wide variations occur in health. The main causes of fast breathing (tachypnoea) are exercise, nervousness and fever; pulmonary, pleuritic and cardiac conditions causing hypoxia; cerebral disturbances, metabolic acidosis and hysterical overbreathing, which latter may give rise to alkalosis and attacks of tetany.

Changes in the character of respiration are discussed in Chapter 7, but one should notice that obstruction in different parts of the respiratory tract may give rise to recognizable varieties of noisy breathing. Obstruction in the nasal passages may cause sniffing or bubbling sounds. Paralysis of the soft palate causes a snoring noise. Obstruction in the region of the larynx causes inspiratory stridor of which one example is the 'whoop' of whooping cough. Obstruction in the trachea may produce growling or rattling noises, as in the 'death rattle', when the lumen is obstructed by mucus. Obstruction in the bronchi may give rise to audible snoring or wheezing noises. Obstruction in the larynx or larger bronchi characteristically gives rise to inspiratory noises, while obstruction in the small bronchi and bronchioles produces expiratory wheezing. The latter is heard in bronchitis and asthma (obstructive airways disease). Alternating periods of cessation of respiration and hyperventilation (Cheyne-Stokes respiration) occur in left heart failure and in various cerebral disturbances. The breathing may be characteristic of diseases quite distinct from those of the respiratory system. Examples of this are stertorous breathing of apoplexy, the hissing expiration of uraemia, and the 'air-hunger' of diabetic coma, which affects both inspiration and expiration.

The odours of alcohol and paraldehyde are easily recognizable in the breath. That of alcohol does not mean that the patient's condition is due to alcoholic intoxication, since alcohol may have been administered as a form of resuscitation. The odour of diabetic ketosis has been described as 'sweet and sickly'; that of uraemia as 'ammoniacal or fishy'; and that of hepatic failure as 'mousy', but one should not rely too far on such delicate distinctions.

THE MENTAL STATE

The examination of the mental state is the equivalent in psychiatry of the physical examination in general medicine. The patient's words should be recorded exactly. With mute or otherwise disturbed patients the 'mental state' examination may be merely a description of behaviour. It is assumed that common psychoneurotic symptoms (anxiety,

phobias, etc.) will have been asked about in the history taking. The important aspects of the mental state examination are:

General observations. Appearance (appropriate, neat, dishevelled, etc.): manner (friendly, unfriendly, suspicious); unusual movements or mannerisms; speech (normal or abnormal words (neologisms)); rapport and 'contact' with the patient. Confusion (of time, place or persons) is an important sign suggesting organic, rather than psychological, disturbance of the brain.

Mood. Elation or depression of mood; appropriateness to the situation.

Thinking. Preoccupations, peculiar 'notions' or ideas, false beliefs (delusions).

Sensations. Unreality feelings (depersonalization); false sensations (hallucinations) commonly of hearing, less commonly vision, taste or smell.

Special attention needs to be paid to the mental state of the *elderly*. The bright and smiling face of an old lady may well mask considerable defects of memory and orientation.

Questions such as 'Where do you live?', 'What place are you now in?', 'What is the day of the week and the date?', 'Who are the other people in the ward or home?' and 'What do they do?' may reveal *disorientation* for place, time and person.

(For further consideration of mental functions see pp. 202-6.)

3

THE GASTROINTESTINAL SYSTEM, ABDOMEN AND PELVIS

The Mouth and Throat — The Abdomen — The Abdominal Viscera — The Genitalia — Some Practical Procedures — Special Investigations of the Gastrointestinal and Biliary Tracts — Examination of Vomit — Examination of Faeces — Intestinal Parasites

THE MOUTH AND THROAT

To examine the mouth and throat, place the patient facing a window or other good source of light, or use a torch.

The teeth

Observe the number of teeth present and look for decay (caries). It is said that lack of teeth may cause indigestion, but many edentulous people suffer no indigestion, whether they wear their dentures or not. The presence of Hutchinson's teeth affords evidence of congenital syphilis, but this is now a very rare finding. In this condition the two central upper permanent incisors are rounded in section and notched at their biting edge. They may also be broader near the gum than at the crown, so as to be peg-shaped. The first permanent molars may be dome-shaped. Much commoner is some notching of the incisors due to the habit of biting cotton or holding hair clips between them, and this must not be taken for Hutchinson's teeth, nor must the broad concave biting edge of ill-formed hypoplastic teeth. In endemic fluorosis chalk-white patches appear on the teeth or they may present a dull unglazed appearance, sometimes with pitting and brown staining (Maldon teeth). Children up to the age of 8 treated with tetracyclines (and children of expectant mothers so treated after the fourteenth week of pregnancy) are at risk of acquiring permanent staining of both the deciduous and permanent teeth. This takes the form of disfiguring horizontal bands, which may be yellow or grey and must not be mistaken for bands of hypoplasia of the enamel due to exanthematous fevers or any serious illness occurring during the development of the crowns. Enlargement of the lower jaw in acromegaly leads to alteration of the bite, so that the lower teeth may close outside the upper ones.

The gums

In *chronic marginal gingivitis*, the gums are retracted, frequently bleed easily and lose their characteristic stippling. Sometimes pus can be squeezed from them (pyorrhœa). *Acute herpetic gingivostomatitis* occurs most commonly in infants and children. Many small vesicles appear on the gums and on the cheeks, palate, tongue and lips. The vesicles rupture to produce shallow ulcers with a yellowish floor and a bright red margin. *Vincent's gingivitis* characteristically destroys the interdental papillae. A thick felted greenish-grey slough is formed, and there is a characteristic odour. In patients exposed to lead compounds, a stippled blue line can often be observed running along the edge of the gum, especially opposite those teeth showing gingivitis. This line must be distinguished from similar discoloration due to a black layer of tartar on the teeth. If a wedge-shaped slip of white paper is inserted between the gum and the tooth, the stippled line of lead poisoning will be rendered more distinct, whereas discoloration due to tartar on the teeth will disappear. The gums may be swollen and spongy in scurvy. Hypertrophy of the gums may occur in pregnancy, and in patients treated for long periods with sodium phenytoin (Epanutin). Haemorrhages may be observed in the buccal mucous membrane in thrombocytopenic purpura and acute leukaemia.

The tongue

Ask the patient to protrude it. Slight *deviation* from the midline is not uncommon and may be due to asymmetry of the jaws. In hemiplegia deviation towards the paralysed side may be found, while in lesions of the hypoglossal nerve or its nucleus there may be fasciculation of the affected side. Later this side may be wasted and deeply grooved—lingual hemiatrophy. The tongue is large in acromegaly and cretinism. *Tremor* of the tongue may be due to nervousness, thyrotoxicosis, delirium tremens, dementia paralytica or parkinsonism.

Next examine the surface of the tongue and note:

1. Its *colour*. Is it pale, red or discoloured? Pallor is seen in severe anaemia. Discoloration is most often due to the ingestion of coloured things, e.g. red wines or coloured sweets.

2. Is it *dry or moist*? The state of the tongue gives some indication of the state of hydration of the body. A dry brown tongue may be found in the later stages of any severe illness, but is found particularly in the later stages of uraemia and acute intestinal obstruction.

3. The presence or absence of *fur*. Furring of the tongue is of little value as an indication of disease. It is found in heavy smokers, mouth breathers, the edentulous and those on soft, milky or otherwise sloppy

diets. A brown fur—the ‘black hairy tongue’—is due to a fungus infection and is of no special significance, though frequently a source of great alarm to its possessor (Plate II). The tongue gives no useful information about the state of the bowels. The tongue of scarlet fever at first shows bright red papillae standing out of a thick white fur. Later the white coat disappears, leaving enlarged papillae on a bright red surface—the ‘strawberry tongue’.

4. The character of the *papillae*. Generalized atrophy of the papillae produces a smooth or bald tongue which is characteristic of pernicious anaemia, but may also sometimes be found in iron-deficiency anaemia, sprue, other gastrointestinal disorders and deficiency states, especially pellagra. In severe cases smoothness may be associated with wrinkling of the mucous membrane, which has then to be distinguished from fissuring of the tongue seen in chronic superficial glossitis due to syphilis, now rare in the UK, and congenital fissuring of the tongue or ‘scrotal tongue’, which is common and of no pathological significance. In chronic superficial glossitis, areas of leukoplakia (whitish opaque areas of thickened epithelium) are separated by intervening smooth and scarred areas and no normal papillae are seen. In congenital fissuring (Plate III) the papillae are normal, but the surface is interrupted by numerous irregular but more or less symmetrical furrows. In median rhomboid glossitis a lozenge-shaped area of loss of papillae and fissuring is seen in the midline anterior to the foramen caecum (Plate III). It may be mistaken for a carcinoma, and must also be distinguished from a lingual thyroid which appears posterior to the foramen caecum. ‘Geographical tongue’ is another harmless anomaly in which the tongue shows localized smooth areas, which change in distribution (Plate II). The ‘false geographical tongue’ with a similar appearance occurs temporarily in children with fever (Plate II).

5. The *undersurface* of the tongue. A small ulcer on the fraenum is sometimes seen in persistent coughing, and particularly in whooping-cough. Sublingual varicosities are common in the elderly.

Inspect the mucous membrane on the inside of the *cheeks*. In the catarrhal stage of measles, before the appearance of the rash, small bluish-white spots, surrounded by a red areola, may be seen opposite the molar teeth. These are known as Koplik’s spots. In the same situation irregular areas of slaty grey or blue pigmentation are seen in Addison’s disease.

Thrush may sometimes be seen on the surface of the buccal mucous membrane, especially in children and in some patients being treated with antibiotics and is common under unclean dentures. It presents the appearance of small white points or patches raised somewhat above the surrounding surface, which is sometimes redder than normal. Patches of thrush are apt to be mistaken for small milk curds, but curds can be

easily detached, while thrush patches can only be removed with difficulty, and when removed tend to leave behind a raw surface.

The palate, fauces and pharynx

Ask the patient to open the mouth wide and, if necessary, introduce a tongue depressor. Note first the general colour of the soft palate, fauces and pharynx; observe any abnormal degree of pallor or redness. The palate and pharynx may be insensitive in hysterical patients. Again, look for any ulcers or mucous patches on the palate, fauces or tonsils. The commonest ulcer seen in the mouth is a small extremely painful, superficial one with a yellow-grey floor and bright red edges—the aphthous ulcer. Mucous patches are slightly raised round or oval areas, covered by pearly grey membrane. They are found in secondary syphilis, as are also superficial, circinate ‘snail-track’ ulcers. Deep clean-cut ulcers with a thick felted grey-green slough on the floor and a characteristic odour occur in Vincent’s angina. Large ragged ulcers or sloughs are seen in agranulocytosis and leukaemia. Ask the patient to say ‘Ah’ thus raising the soft palate and increasing visibility. Look carefully at the *tonsils*, noting any enlargement. Yellowish or greyish points or patches may sometimes be seen on their surface. See if these can be wiped off, leaving a sound surface, as is the case with accumulated follicular secretion, or whether removal leaves a raw surface, as happens with the false membrane of diphtheria. Note also if the soft palate and uvula show any similar spots or patches. In glandular fever pinhead petechiae are often seen on the hard palate and a membrane may be seen on the fauces. Petechiae may also be seen on the mouth and throat in any form of thrombocytopenia. The membrane of diphtheria is found characteristically on the mucous membrane of the fauces, as well as on the tonsils. Bacteriological examination of a throat swab is essential when there is a suspicion of diphtheria.

Next look at the *pharynx*. The presence upon its surface of a number of flat adenoid swellings, somewhat like sago grains, is so common as to be almost a normal appearance. In chronic pharyngitis these are much increased. A few dilated venules can also be frequently observed. Notice any pus or excess of mucus on the surface and the existence of any ulceration. In retropharyngeal abscess the posterior wall of the pharynx is bulged inwards. Sometimes this can be more easily made out by palpation.

The breath

Bad teeth, gum or mucous membrane ulceration or retention and decomposition of secretion in the follicles of enlarged tonsils are the commonest sources of offensiveness in the mouth.

It is important to learn to recognize the characteristic odours of

ketosis, uraemia and hepatic failure. In ketosis the breath smells of acetone, in uraemia there is a fishy or ammoniacal odour, and the odour of hepatic failure has been described as 'mousy'.

In suppurative conditions of the lung the breath may have a putrid smell, while the odour of bronchiectasis has been compared to that of apple blossom with an *arrière-goût* of stale faeces. Paraldehyde and alcohol also impart their characteristic odor to the breath.

THE ABDOMEN

The patient should be lying flat on his back in a good light. The abdomen is exposed by turning down all the bedclothes except the inner sheet. The clothing should then be drawn up and, lastly, the sheet folded down a little above the level of the pubes. These details are of special importance in examining female patients. Before beginning the examination of the abdomen, ensure that the bladder is empty. The abdomen is usually best examined with the patient fully recumbent, with not more than one pillow and with the arms at the side.

Inspection of abdomen

First look at the general shape of the abdomen. Is it of normal fullness, or protruberant, or is it sunken? General fullness may be due to 'fat, fluid, flatus, or faeces' to which aphorism may be added 'fetus' in the case of women. A large ovarian cyst or an enormous spleen or liver may also produce enlargement, but this is asymmetrical. In all cases of general abdominal swelling one should measure the circumference at the point of maximum distension. Observe in which zone local bulging is situated. Lastly, note if there is any movement to be seen in the swelling, either along with or independently of respiration.

Pulsation in the epigastric region may be noticed. Usually it is simply transmitted from the aorta in a thin and nervous person. Occasionally it is due to a hyperkinetic right ventricle in a patient with right heart strain, or to transmission from the aorta by a growth, e.g. carcinoma of the stomach. More rarely it is due to an aneurysm of the abdominal aorta, in which case an expansile swelling will be felt, or one may simply notice an increase in the width of the aorta. Some enlargement of the aorta is not uncommon in old people.

The *movements of the abdominal walls* should be studied. Normally they bulge during inspiration and fall in during the expiration. Paralysis of the diaphragm produces the opposite picture; sometimes the paralysis is unilateral, in which case one side of the abdomen will move naturally. Absence of movement of the abdominal walls is a sign of peritonitis.

Severe intestinal obstruction produces tympanitic abdominal distension and sometimes visible peristaltic waves. The most extreme

degrees of distension are seen in obstruction of the distal parts of the large intestine and chiefly affect the periphery of the abdomen. In obstruction at the ileocaecal valve, the distended coils of the small intestine tend to stand out in the centre of the abdomen in a 'ladder pattern', but small intestinal peristalsis may sometimes be seen in old persons with thin abdominal walls and in the presence of divarication of the recti in persons who are otherwise healthy. In pyloric obstruction, the dilated stomach forms a prominent swelling in the abdomen, and on gently shaking the patient a splashing noise is produced (which may also occur in healthy patients after a meal and in other forms of intestinal obstruction). The swelling may be seen in the upper abdomen in acute obstruction but with large dilated stomachs it may be in the mid or lower abdomen. Contractions in the stomach run from left to right. They are of particular importance in congenital pyloric stenosis of infants, when they may be the only diagnostic sign. They may be easier to see if the infant is given a bottle or an adult a drink of soda-water.

Next look at the *surface of the abdomen*. In great distension the surface is smooth and glossy. *Striae* (white or lilac lines in the epidermis) indicate a recent change in the size of the abdomen, and thus are found in pregnancy, ascites and wasting diseases. Large, livid purplish striae are characteristic of Cushing's syndrome, but minor degrees may sometimes occur in obese people who are otherwise healthy. Note any *distension of the surface veins*, and try to ascertain in what direction the blood is flowing. In obstruction of the inferior vena cava (Plate IV) the inferior epigastric veins are full from the establishment of a collateral circulation. In such cases also a large *lateral vein* can be seen running up about the midaxillary line, establishing a communication with the tributaries of the superior vena cava, so that the blood is flowing upwards. Distended veins may also be seen on the abdomen in portal obstruction (Plate IV). Very rarely in this condition a number of distended veins may be seen radiating from the umbilicus. To this appearance the term 'caput Medusae' has been applied. It is due to establishment of a connection between the portal and parietal veins by means of the round ligament. Small distended venules in the region of the costal margin are of no significance. *Pigmentation* of the abdominal wall is sometimes important. Along the middle line it forms the *linea nigra*—one of the signs of pregnancy. Note the appearance of the *umbilicus*. Is it depressed, level with the surface, bulging or everted as in gross ascites? Lastly, one should never omit to look at the usual sites for any evidence of hernia.

Palpation of the abdomen

The patient should lie flat on his back but relaxation is sometimes aided by flexing the hips and knees. He should be asked to keep the

mouth open and to breathe quietly; his attention may be diverted by conversation. The observer must sit or kneel beside the patient, in order to get his forearm into the horizontal position. Ordinary palpation is performed with one hand only. The hand must be warm. In order to gain the confidence of the patient, the hand should be allowed to rest for a moment on the surface of the abdomen before palpation is actually begun. Poking with the fingertips should be avoided at all costs, the best movement being a gentle one from the metacarpophalangeal joints, with the hand flat on the abdominal wall. Organs and masses in the upper abdomen are best felt against the radial border of the index finger as they descend in inspiration, at which time the hand may be moved gently upwards, as if to 'meet' them. To examine lateral regions of the abdomen, bimanual palpation is carried out. The physician sits or kneels by the bedside. One hand is placed posteriorly in the loin. The other is placed over the abdominal wall in front. The posterior wall is then pushed up against the hand in front, so that any structure lying between the two hands can be distinctly felt as the patient breathes in. This procedure is of special value in the examination of the kidneys (p. 48).

Begin by a systematic very gentle palpation of the whole abdomen, noting any local or general rigidity or any marked tenderness. In this way the patient's confidence is gained and the later deep palpation rendered easier. It is wise to ask the patient whether any area is tender and, if so, to come to that area last. Normally the abdomen has an elastic or doughy feeling only to be learnt by experience. In disease the resistance may be increased. General peritonitis may produce rigidity from a reflex contraction of the muscles of the abdominal wall. Local rigidity may be due to localized peritonitis. Palpation of the normal abdomen is painless. If tenderness is elicited, its exact extent and point of maximum intensity should be noted.

Feel next for any unusual swellings in the abdomen. These if present may be enlarged organs, the characteristics of which will be described later, or masses of other kinds. If you think that you feel something abnormal, consider first whether it could be a normal structure. Faeces in the colon, a bulky caecum in the right iliac fossa, the descending colon in the left iliac fossa, a full bladder or an abnormally mobile kidney almost anywhere in the abdomen are the most common causes of error.

If you do feel a mass, note:

1. Its position in the abdomen. Does it extend under the costal margin or into the pelvis? Or does it arise from the pelvis?
2. Its size and character. Is it hard, soft or cystic? Is it fixed or movable? Does it move with respiration?
3. Is it attached to any particular organ? Is it attached to the anterior abdominal wall?

Percussion of the abdomen

This should be carried out in the same manner as will be described for the chest, but particular care should be taken to percuss *lightly*. Percussion of the normal abdomen yields a tympanitic note throughout, except in the regions of liver and splenic dullness, or over a full bladder. Enlargement of these organs may sometimes be confirmed by percussion. The splenic dullness may be increased when the spleen is ruptured. The liver dullness may be absent when there is gas or air in the peritoneal cavity. Such absence is a sign of perforation of a viscus, usually of perforation of a peptic ulcer. The liver dullness is reduced in severe emphysema and in the presence of a right pneumothorax.

Free fluid in the peritoneum (*ascites*) should be suspected if there is generalized diffuse enlargement of the abdomen; it is unwise to diagnose fluid if this is not present. It may also be possible to demonstrate 'shifting dullness'. If the patient is turned over on his side and time given for the intestines to float up, the uppermost flank becomes resonant, while the height of the dullness on the lower side rises.

The 'fluid thrill' is another physical sign of fluid in the peritoneum. To elicit this sign, the patient is laid on his back, one hand is placed over the lumbar region on one side and the opposite side is flicked or tapped with the fingers of the other hand. A distinct impact will be felt to pass from one hand to the other. A similar impulse can be transmitted through a fat abdominal wall and so it is necessary for an assistant to place the edge of his hand firmly in the middle line of the abdomen while the tapping is performed. This damps down vibrations transmitted by the wall. A fluid thrill can only be expected when the amount of fluid is large and it is under tension.

Fat is distinguished by taking the abdominal wall between the hands and pinching it up; *gas* by the results of percussion. Of *new growths*, ovarian cyst is probably the most liable to be mistaken for ascites. An ovarian cyst, however, causes an anteroposterior bulging of the abdomen, while in ascites the bulging is mainly lateral. In ovarian cysts the dullness is central and does not change with the position of the patient: in ascites the chief dullness is in the flanks and it shifts when the patient is moved: the umbilicus is flat or bulges out in ascites, while in ovarian tumours it is drawn upwards: the 'slit' in the umbilicus is usually transverse in ascites and vertical in ovarian cyst.

Auscultation of the abdomen

When one listens to the normal abdomen various tinkling and gurgling sounds are audible with occasional loud borborygmi. However, in cases of general peritonitis or intestinal ileus these sounds may be reduced or absent. The 'silent abdomen' is a valuable sign of an inactive bowel.

Excessive sounds may be heard in intestinal obstruction, sometimes coinciding with waves of pain.

Systolic murmurs may be heard over partially obstructed mesenteric, renal, splenic or femoral arteries and over aneurysms; and a continuous venous hum over anastomotic veins or varices in portal obstruction.

THE ABDOMINAL VISCERA

The liver

Palpation of the liver

Feel for the lower edge. To do this, place the hand flat on the abdomen, its edge towards the costal margin and to the outer side of the rectus muscle, thus avoiding the upper septum of the rectus sheath, which may be mistaken for the lower edge of the liver. Depress the edge of the hand slightly so as to push up a fold of skin and ask the patient to take a deep breath. If the edge of the liver is palpable, it will be felt to ride under the edge of the hand. Trial must be made at different levels working from below upwards before it is decided that the edge cannot be felt. The edge of the liver can sometimes be felt in health and is frequently palpable in patients with emphysema. The character of the surface should be made out; whether it is soft, smooth and tender, as in heart failure; firm or nodular as in cirrhosis; or hard and coarsely irregular, as in secondary carcinoma. In rare cases of tricuspid regurgitation it may be felt to pulsate.

Percussion of the liver

Use fairly heavy percussion. Begin high up at about the second rib, to get a good lung note, and percuss down until impairment is detected. The upper limit of liver dullness forms an almost horizontal line around the chest. To define the lower edge of the liver, use very light percussion and pass upwards.

The gall bladder

The gall bladder cannot be felt unless distended, when it may form a smooth pear-shaped swelling, situated just to the outer edge of the right rectus muscle. It can be moved freely from side to side round a point opposite to the ninth costal cartilage. It moves with respiration.

If present, tenderness of the gall bladder can be elicited by placing the hand beneath the costal margin in the right hypochondrium. The patient is told to take a deep breath and at the same time the hand is moved upwards underneath the costal margin. As the diaphragm descends, the

gall bladder is driven against the fingers and if it is tender the breath is arrested with a gasp. This is spoken of as *Murphy's sign*.

The spleen

Palpation of the spleen

The normal spleen is not palpable. A palpable spleen is at least twice the normal size and it is never safe to diagnose enlargement of the spleen unless it *is* palpable.

Feel for the spleen standing on the right side of the patient. Place the flat of the left hand over the edge of the costal margin and firmly bring the ribs and lateral abdominal wall medially as the patient breathes in. Then start palpating with the right hand. (Large spleens are missed by starting palpation too near the costal margin.) The edge of the enlarged organ will be felt against the fingers of the right hand. Sometimes in the case of minor degrees of splenomegaly the organ is best felt if the patient rolls over, half on to his right side towards the examiner.

The edge of the spleen is sharp and usually quite smooth. A notch may be felt if the spleen is considerably enlarged. It is important to note that the spleen enlarges downwards and towards the right iliac fossa (Fig. 3).

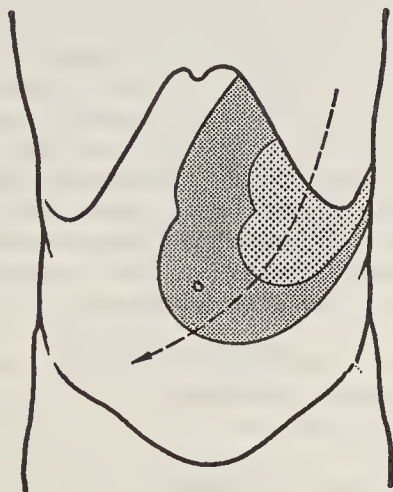


Fig. 3. The direction of enlargement of the spleen.

Auscultation of the spleen

Friction sounds may be heard in perisplenitis and over the surface of splenic infarcts.

The kidneys

Palpation of the kidneys

The patient must lie on his back with the lumbar region flat and not arched forward. Place one hand upon and below the last rib behind, the other below the costal margin in front. The posterior hand should press the loin forwards, while the other hand pushes the anterior abdominal wall backwards, upwards and inwards. If the abdominal muscles are relaxed and the patient takes a deep sighing inspiration, the kidney, if enlarged or displaced, can be felt to move downwards and then on expiration upwards. If the kidney is enlarged, it is possible to trap it and so momentarily to prevent it from returning to its normal position. In healthy thin persons the lower pole of the right kidney is often palpable, the left seldom so.

A movable right kidney is apt to be mistaken for a distended gall bladder, and vice versa. Points of distinction are that a distended gall bladder can often be seen on inspection; it can also be momentarily pushed back from the abdominal wall but tends to spring forward again; it is therefore always in evidence. A movable kidney, however, often disappears on expiration and can only with difficulty be found again on inspiration. Further, only the kidney is bimanually palpable.

An enlarged left kidney may be mistaken for the spleen. The points of distinction are that (a) the spleen has a sharp edge in which a notch can often be felt but the edge of the kidney is always rounded and has no notch; (b) the fingers can usually be passed between the upper end of a renal tumour and the ribs but not between the ribs and a splenic tumour; (c) a renal tumour is usually 'bimanually palpable', that is, can be moved forwards and backwards between one hand in the loin behind and the other on the anterior abdominal wall, but an enlarged spleen is not 'bimanually palpable'; and (d) the colonic resonance may be detected passing in front of a renal tumour, but not in the case of a splenic tumour.

An enlarged kidney tends to bulge forwards. Perinephric abscesses and extravasations bulge backwards.

The bladder

In normal health the bladder cannot be felt. If it is greatly distended with urine it can be felt above the symphysis pubis extending towards or above the umbilicus. It may remain central in position or fall to the right or left side. On percussion there will be dullness over a distended bladder.

THE GENITALIA

The male genitalia

The patient should lie on his back, with his legs apart. Note the pubic hair, the appearance and size of the penis, the presence or absence of a prepuce and the site of the external urethral orifice. Inspect the scrotum for redness, swelling, oedema or distension. If any fullness or swelling is present observe whether it extends into the groin. A hernia is shown by an impulse on coughing which is often better seen than felt.

Palpation must be carried out gently. The penis should be palpated for any induration of the corpora cavernosa, and for any induration, swelling or ulcer of the glans. One should establish that the prepuce can be readily retracted and then drawn forwards over the glans, thus excluding phimosis and paraphimosis. The lips of the external urethral orifice should be separated to inspect its lumen.

Before one examines the scrotum the presence of any tenderness or enlargement of the inguinal lymph nodes should be noted, as should the pulsation in the femoral arteries. The size and consistency of each testis and epididymis should be noted. A scrotal swelling, which feels like a bag of worms, especially when the patient stands up, is likely to be *varicocele*. One which extends into the groin, as determined by feeling the spermatic cord on each side and by the presence of an impulse on coughing, is likely to be an *inguinoscrotal hernia*. A diffuse, pear-shaped, cystic swelling, which may be either tense or lax, obscuring and surrounding the testicle will be a *hydrocele of the tunica vaginalis*. In early cases such a swelling will faintly transmit the light of a pocket torch. For further information about scrotal swellings, textbooks of surgery must be consulted.

The internal male genitalia (the prostatic gland and seminal vesicles) are examined per rectum (p. 51).

The female genitalia

It goes without saying that any examination of the female genitalia must be performed with gentleness and consideration. Good lighting, a firm examination couch and a chaperone are essential. Light-weight disposable gloves and lubricant jelly will be needed: and a vaginal speculum, an Ayres spatula for cervical cytology, standard culture swabs and Stuart's transport medium should be available.

The patient should be requested to empty her bladder immediately before the examination. In the first place she should be asked to lie in the left lateral position with the knees flexed towards the abdomen; at a

later stage she may be asked to turn to the dorsal position for bimanual palpation of the adnexa.

Inspection and vaginal examination

Careful observation is made of the external genitalia, perineum, anus and inner thigh regions. Using the index finger and thumb the labia minora are then separated for inspection of the clitoris, urethra and introitus. If the hymen is intact, no further digital examination is attempted unless under anaesthesia, but bimanual palpation of the upper genital tract is undertaken per rectum. If the hymen has been ruptured, a speculum is inserted and appropriate swabs may be taken from the urethra, the vaginal vault of the external os of the cervix. After inspection of the cervix, a cytological smear is obtained by placing the Ayres spatula into the external os of the cervix and rotating it through 360°. All women over the age of 30 who have had regular intercourse or a pregnancy should have this investigation at not more than 2-year intervals. In this way more carcinomas of the body of the uterus and the cervix should be discovered at a treatable stage. Inspection is completed by asking the patient to cough or bear down in order to demonstrate genital prolapse or stress incontinence of urine.

Bimanual digital examination

Initially the right index finger alone is inserted into the vagina, pressing gently towards the perineum. When the pelvic floor muscles are felt to relax, both index finger and middle fingers can be inserted more readily and the thumb raised forwards over the symphysis, but held away from both urethra and clitoris. The left hand is placed carefully on the lower abdomen suprapubically and gentle downward pressure applied. This not only distracts the patient from the anxiety of the vaginal examination, but also serves to push the uterus down towards the fingers of the right hand. Bimanual palpation of the uterus is a continuation of cervical assessment and is best achieved by approximating the fingers of both hands to outline the contours of uterus and ovaries. Throughout the examination, the patient's facial expression should be observed to note any pain elicited, in particular on moving the cervix or palpating the adnexa. The most satisfactory deep palpation of the pelvic floor and pouch of Douglas region is performed by inserting the index finger into the vagina and simultaneously the middle finger in the rectum.

For the findings on gynaecological examination and their interpretation, specialized textbooks must be consulted. This examination must sometimes, however, be considered an essential part of a general examination, as an aid for instance to elucidating the nature of an abdominal swelling or the cause of ascites.

SOME PRACTICAL PROCEDURES

Rectal examination

Place the patient in a good light and curled up on his left side, that is, in the left lateral position. Draw aside the buttocks and inspect the region of the anus, noting the presence of any eruption, of external haemorrhoids and of fissures or fistulae. Fit a disposable finger-stall or glove and smear it with petroleum jelly. Massage the anus for a moment with the finger and then press gently with the pulp of the finger till it enters the anus, directing it slightly forwards at first. Patients often groan or otherwise object to rectal examination, though it is almost painless in a healthy person. Real pain is usually due to an acute anal fissure which may be difficult to see.

Once the anal canal is passed, direct the finger slightly backwards and upwards, asking the patient to bear down a little at the same time. The finger can then be swept round and the whole inner surface of the rectum explored.

In the male the prostate will be felt projecting into the rectum, and above it the trigone of the bladder flanked by the seminal vesicles which in health may not be palpable; below is the membranous urethra. In the female the cervix will be felt projecting back in the form of a firm rounded swelling. Feel the mucous membrane for polyps, ulcers and tumours.

Remember that haemorrhoids are not palpable unless they are thrombosed. The presence of scybala or foreign bodies can be determined. If the lymphatic glands which lie in the hollow of the sacrum are enlarged, they may be felt. If secondary malignant deposits or an abscess are present in the rectovesical pouch, the mass will be palpable through the wall of the rectum. On withdrawing the finger, examine the finger-stall for the presence of mucus, blood or melaena. Fragments of stool adhering to the finger-stall can be tested for occult blood (p. 64).

Proctoscopy and sigmoidoscopy

If rectal examination is negative and there is reason to suspect abnormality near the anus, the anal canal and lower 7 cm of the rectum should be examined with the proctoscope. Place the patient in the position described for rectal examination, and pass the warmed, lubricated instrument carefully to its full depth. Remove the obturator and inspect the mucous membrane as the instrument is slowly withdrawn. In this manner haemorrhoids may be seen or the nature of a palpable abnormality directly ascertained.

It is often necessary to examine the rectum and colon more fully than is possible by proctoscopy, and in such cases the sigmoidoscope is

employed. Sigmoidoscopy requires skill and experience. In accomplished hands the instrument can be passed for 20 cm and a further 4 cm or so of the colon is visible beyond this. The procedure causes very little discomfort and anaesthesia is unnecessary and undesirable.

Sigmoidoscopy is particularly useful in the differential diagnosis of diarrhoea of colonic origin. Proctitis, polyps and carcinomas may be seen, and biopsies taken.

In suspected amoebic dysentery, the mucous membrane may be inspected, and portions of mucus or scrapings from ulcers may be removed, mounted in saline on slides, and examined microscopically for amoebae and cysts. When the sigmoidoscope is used for this purpose, it should be lubricated with a non-greasy preparation, such as mucilage, or droplets of oil will interfere with the microscopical examination.

Aspiration of peritoneal fluid

This is undertaken for diagnostic and therapeutic purposes. It is essential first to make sure that the bladder is empty, and if there is any doubt a catheter should be passed before the aspiration is attempted.

The patient should be lying flat or propped up at an angle of 45°. A binder or many-tailed bandage should be placed in position around the patient's back before the aspiration is begun. The aspiration is usually performed in the right iliac fossa, a little outside the mid point of a line drawn from the umbilicus to the anterior superior spine.

With suitably sterile precautions, the skin at the point chosen should be infiltrated with local anaesthetic, and then the anaesthetic should be injected down to the parietal peritoneum. If the puncture is made simply for diagnostic purposes, a 10 ml syringe and a suitable needle can be used.

If it is intended to drain the peritoneum, a trochar and flanged cannula (which can be fixed to the skin with adhesive tape) should be employed. It is wise to tie the rubber tubing (which is used to drain the fluid into a bottle) on to the flanged cannula, then to stretch the tubing over the end of the cannula and to insert the trochar through the rubber tubing. This saves soiling of the bedclothes when the trochar is withdrawn. A tiny incision should be made in the anaesthetized area of skin, and then the trochar and cannula should be inserted. A resistance is felt as the trochar perforates the parietal peritoneum. The trochar should then be withdrawn, and the fluid should drain into the bottle. The rate of flow, which should not be too fast, can be controlled by means of a clip on the rubber tubing. When the aspiration is complete, the cannula should be withdrawn. The puncture can be sealed with collodion and a dry dressing applied. Therapeutic drainage should, however, be avoided if possible; diuretics are preferable.

The fluid can be examined bacteriologically, chemically and for

cytology. Transudates such as occur in heart failure, cirrhosis and nephrosis normally have a specific gravity less than 1018 and a protein content under 2.5 g/litre. Exudates, occurring in tuberculous peritonitis or in the presence of secondary deposits, usually have a specific gravity above 1018 and more than 2.5 g of protein/litre. The distinction, however is somewhat unreliable. Tubercle bacilli may be demonstrated in the fluid in tuberculous peritonitis; blood-stained fluid strongly suggests metastases. Malignant cells may also be demonstrated in the latter condition and in malignant ascites the fluid recurs rapidly after paracentesis.

Exploration of liver for liver abscess

Liver abscesses due to *Entamoeba histolytica* are nearly always found in the right lobe of the liver. When liver scans are available, the presence and position of an abscess should be demonstrated by this means; but in any ill patient with signs pointing to this diagnosis, exploration for both diagnostic and therapeutic purposes should not be long delayed. The method is as follows. The main danger is that the needle will enter the inferior vena cava. Puncture of the intrahepatic branches of the portal vein usually does no harm. It has been shown that in a man with a chest of 80 cm circumference, the inferior vena cava is nowhere less than 10 cm from the surface. A needle of wide enough bore to admit thick pus (a stout old-fashioned lumbar puncture needle is suitable) is selected and a piece of adhesive tape is wound around it 9 cm from its point. After anaesthetization of the skin and subcutaneous tissues, the needle is entered either at the site of maximum tenderness or in the eighth, ninth or tenth space in the right axilla, and passed laterally, i.e. towards the patient's opposite side and slightly upwards towards the patient's head, to a maximum depth of 9 cm. By this means the whole of the right lobe of the liver can be explored. The pus in a liver abscess may be thick. Thus, as already mentioned, a stout needle must be used, and fairly strong suction maintained with the syringe.

If pus is encountered, it is usual to remove as much as possible with the aid of a two-way syringe. As the pus is removed, it may be replaced by air either until the patient complains of pain in the liver or shoulder, or until half of the volume of pus removed has been replaced by air. If the patient is then X-rayed in several positions the exact site and size of the abscess can be determined, and the effect of treatment on its size can be followed.

SPECIAL INVESTIGATIONS OF THE GASTROINTESTINAL AND BILIARY TRACTS

Under this heading methods of examining the stomach, gall bladder and intestine by intubation and radiology will be described.

Intubation

Intubation is used more for therapeutic than diagnostic purposes, but is a necessary preliminary to studies of gastric acid production.

The original Ryle's rubber tube has been replaced by disposable plastic ones, which are sterilized by gamma radiation and supplied in sealed packs. The usual size is 16 French gauge. The blind end is weighted with metal and is thus radiopaque; and there are a number of small perforations a few centimetres from the tip. Marks on the tubing show the expected position of the tip, when each mark is opposite the teeth: but when these tubes are used for diagnostic purposes, the position of the tip should always be confirmed by screening. For gastric acid studies the tip should be in the most dependent portion of the stomach.

The tube may be placed on the back of the tongue and the patient then swallows it like a pill perhaps with the aid of a little water: or the lubricated tube may be passed through the nasal passages and then swallowed. When necessary the tip can usually be induced to pass into the duodenum. The patient should lie on his right side, tilted very slightly forwards. Experts in duodenal intubation make use of various further manipulations and claim to get the tip into the duodenum regularly and speedily. Once more the position of the tip should be confirmed by screening.

Studies on gastric acid production

Differences in gastric acid secretion can readily be demonstrated between groups of patients with gastric ulceration, patients with duodenal ulceration and normal subjects, but the wide range of secretion in each group means that differences between individuals are of little significance. For this reason gastric acid studies are no longer used in differential diagnosis of dyspepsia and 'test meals' have become obsolete.

Measurements of gastric acidity are used:

1. To confirm a diagnosis of pernicious anaemia where facilities for Schilling tests, serum vitamin B12 and serum folate levels are not available. Patients with pernicious anaemia show achlorhydria (defined as the failure to lower the pH of the gastric juice below 6 after a maximum stimulus to secretion).

2. To detect the gross hypersecretion of acid, particularly in the basal secretion, in the rare Zollinger-Ellison syndrome.

3. By some surgeons as an aid to deciding the type of operation to be performed in patients with peptic ulceration.

As with many modern investigations these studies are only of value if performed by experienced persons who do them regularly. The figures obtained may vary considerably according to the positioning of the tip of the tube and method of suction employed.

The patient should neither eat, drink nor smoke after midnight. After the position of the tip has been confirmed by screening, all resting fluid should be removed by gentle manual suction with a syringe. The patient

should then lie on his left side or sit up, whichever is more comfortable. Continuous mechanical suction has been recommended, but experts in the method prefer constant manual suction performed gently with a 20 ml syringe.

It is usual to measure the basal acid output (BAO) and the maximum acid output (MAO), though the second is the more informative.

Basal acid output (BAO). This is usually measured for 1 hour. If the resting fluid is removed completely at the onset, the basal secretion in normal persons is a few millilitres per hour, containing up to 10 μ Eq of hydrochloric acid. In the rare Zollinger–Ellison syndrome the volume is likely to be much higher and the acid produced may be as high as 500 μ Eq/hour.

Maximum acid output (MAO). The synthetic pentapeptide of the hormone gastrin ‘pentagastrin’ has replaced histamine as the agent to produce maximum acid secretion, because it is readily available and, unlike histamine, is virtually free from side effects.

After the basal secretion has been measured for 1 hour a dose of 0.6 mg/kg body weight of pentagastrin is given by subcutaneous or intramuscular injection. This causes a maximum acid secretion, not increased by any further increase in the dose of stimulant. After the injection the juice is collected, preferably by constant manual suction, for 1 hour. The total acid secreted in 1 hour is the MAO. In normal subjects this may reach a maximum of 24 μ Eq/hour in males and 20 μ Eq/hour in females. Values of from 20 to 110 μ Eq/hour are found in patients with duodenal ulceration and much higher figures in the Zollinger–Ellison syndrome.

The insulin test. An injection of insulin produces hypoglycaemia, which stimulates the nucleus of the vagus in the brain stem. Impulses then run down an intact vagal nerve, which stimulate the production of a small amount of acid in the stomach, but this will not happen after a complete vagotomy. Measurement of gastric acid production after an injection of insulin can thus be used to test the completeness of a vagotomy; this investigation is particularly helpful when a patient develops a recurrence after the operation of vagotomy for duodenal ulceration.

Small intestine biopsy

Biopsy of the small intestinal mucosa can be performed using a spring-loaded capsule (the Crosby capsule). This capsule is about 1.5 cm long and 7 mm in diameter. It contains a cutting blade which excises a small piece of intestinal mucosa when negative suction is applied to the thin plastic tube to which it is attached. The capsule is swallowed, and a

small amount of radiopaque dye is injected, to check that it has passed through the pylorus. After the biopsy has been taken, the capsule is pulled up and the specimen placed on a small piece of card in a preservative solution. It should be examined immediately by the pathologist under a low-power microscope, to assess the general appearance of the villi.

This procedure must always be preceded by a barium meal and follow-through examination, as the technique might be dangerous if the capsule were to lodge in a duodenal or jejunal diverticulum.

Small-intestinal biopsy is of particular importance in the diagnosis of the malabsorption syndrome, where a flat mucosa is seen in place of the usual multiple villi.

Radiography of the abdomen

Plain radiographs of the abdomen with the patient standing upright are of great value in cases of suspected peritonitis due to rupture of a hollow viscus, when gas may be seen under the diaphragm; in suspected intestinal obstruction, when fluid levels may be seen in the intestine; and in acute colitis, to aid in the diagnosis, to assess the degree of colonic dilatation and to diagnose perforation. Films taken in the erect and prone positions may also be useful in assessing the size of organs, in detecting calcified objects (e.g. radiopaque gall stones, calcification in the pancreas, calcified hydatid cysts); and in observing the distribution of gas and faecal shadows in various other conditions.

Barium studies are often of the greatest value, but for details of their performance the student must consult special textbooks.

For the barium meal the patient swallows a suspension of radiopaque barium sulphate, while the radiologist observes its passage on the fluorescent screen, or on the TV monitor of an image-intensifier. Films are taken to provide a permanent record of any abnormality discovered, but, as in all barium examinations of the alimentary tract, screening is important. The barium meal is principally used in the diagnosis of gastric and duodenal ulcer and of gastric carcinoma.

The barium filled crater of a chronic *gastric ulcer* may be seen in the stomach as a projection from the wall ('profile' view) or as a rounded deposit ('*en face*' view) with in either case mucosal folds radiating towards the crater. A *duodenal ulcer* is usually seen *en face* with a stellate appearance of the mucosal folds. Often no definite crater is seen, but the cap is deformed as a result of scarring, most classically to produce a trefoil deformity, sometimes with pseudo-diverticulae (Plate XX). There is often an increased amount of resting juice due to some pyloric obstruction, and a grossly enlarged stomach with delay in emptying is found when pyloroduodenal stenosis is present. Persistent

pylorospasm and irritability of the cap which is thus difficult to fill with barium, leads to a suspicion of duodenal ulceration.

Polypoid *gastric carcinomas* cause filling-defects in the barium-filled organ. Cancerous ulcers may be difficult to differentiate from simple ulcers, and the radiologist pays particular attention to the mucosal folds and mobility of the wall in the region of the ulcer. The linitis plastica type of growth gives a rigid conical shape to the stomach with absence of peristalsis and no ulceration. Carcinomas involving the cardia and pylorus cause obstruction and, if small, may be difficult to differentiate from simple lesions (Plate XIX).

The small intestine may be studied by taking films of the abdomen at intervals after the barium meal. Abnormalities in small-gut pattern—dilatation, narrowing, increase in transverse barring or flocculation—and in the transit time to the colon, may be demonstrated in *malabsorption states*. Areas of narrowing with proximal dilatation, fistulae and mucosal abnormalities may be produced by *Crohn's disease*. Many radiologists prefer to give smaller quantities of a non-flocculating barium suspension, either by mouth or via a duodenal tube—‘the small bowel enema’.

In the *barium enema* the suspension is introduced into the rectum as an enema, and manipulated round the colon to the caecum. By this means, *obstruction to the colon, neoplasm, diverticular disease* and other abnormalities can be recognized (Plate XXI).

Cholecystography and cholangiography

Cholecystography depends on the fact that certain iodine-containing compounds are excreted by the liver and concentrated in the bile and so render the gall bladder radiopaque.

The substance most used in oral cholecystography is Telepaque (iopanoic acid).

After a preliminary radiograph has been taken, to see if radiopaque gall stones are present, a dose of the preparation suitable for the patient's weight is given by mouth after the patient's evening meal, which should be light and free from fat. Several radiographs are taken 12–16 hours later. If the gall bladder is seen, the patient is given a fatty meal and its emptying is observed.

Failure to demonstrate the gall bladder may be due to the fact that the dye was vomited or not absorbed on account of diarrhoea or other cause, that hepatic function is impaired—it is generally useless to attempt cholecystography in a jaundiced patient—or that disease of the gall bladder is present. Deformities of the gall bladder shadow due to structural changes and calculi, whether opaque or not to X-rays, may thus be demonstrated.

Cholangiography can be performed by the intravenous administration of Biligrafin (a compound of triiodo-benzoic acid); 20 ml of 20% Biligrafin are injected intravenously. A film is taken 20 minutes later and then serial films at 10-minute intervals. In successful cases the biliary ducts are outlined, so that patency of the ducts and the site of extra-hepatic obstructions can be demonstrated. The method is particularly valuable to show strictures or calculi in patients whose gall bladders have been removed. It will not, however, be successful in patients with more than slight degrees of jaundice. Increasing the dose of oral preparations does not usually produce denser shadows of the gall bladder or ducts. Intravenous cholangiography is best performed immediately the cholecystogram has been found to fail, that is, 16 hours after the oral Telepaque.

For a description of transhepatic cholangiography and operative cholangiography larger works must be consulted.

Endoscopy

Oesophagus, stomach and duodenum. Rigid gastroscopes have been replaced by flexible fiberoptic instruments. These have many advantages. The examination can be carried out rapidly and safely, with the aid of local anaesthesia alone, and under outpatient conditions. By the use of suitable forward-viewing and side-viewing instruments it is possible to see the whole of the oesophagus, the stomach and the duodenum. It is thus possible to inspect the orifices of the bile and pancreatic ducts, and to cannulate them. The instruments carry a channel through which biopsy forceps or a brush can be introduced to obtain specimens for cytological examination. Most instruments are fitted with a proximal camera, so that permanent records of any abnormality can be obtained.

These examinations should be regarded as ancillary to radiographic ones, which are usually performed first.

Colon. Fiberoptic instruments known as colonoscopes are also available for the examination of the whole colon. With their aid the mucosa of the whole colon can be inspected, biopsies can be taken and occasionally pedunculated polyps can be removed without a laparotomy. All fiberoptic instruments are expensive however and colonoscopy is unlikely to replace sigmoidoscopy for routine purposes.

Liver function tests

The liver is remarkable for the number and variety of its functions. Numerous tests of individual functions have been used. No single test

can give a composite picture of liver function. The more important tests are described below.

Tests related to bilirubin metabolism

Plasma bilirubin. Bilirubin can circulate in the blood in two forms—*unconjugated* (bilirubin which has not passed through the liver cells) and *conjugated* (bilirubin which has been conjugated to form a glucuronide in the liver cells, is water-soluble and so can pass from the blood stream into the urine). Normal plasma contains up to 1 mg/100 ml of bilirubin (17 μ mol/litre), almost all unconjugated (though a few persons with hereditary non-haemolytic disorders of bilirubin metabolism may show values of up to 2 or 3 mg/100 ml).

In haemolytic jaundice the unconjugated bilirubin rises to at most 5 or 6 mg/100 ml. In both intra- and extrahepatic obstruction, the total plasma bilirubin may at times be very much higher (up to 30 mg/100 ml or more) with a high proportion of conjugated bilirubin, which is excreted in the urine.

The plasma bilirubin is most useful in demonstrating levels of jaundice below 2 mg/100 ml which do not cause a visible yellow colour in the skin and conjunctivae, in demonstrating the haemolytic nature of a jaundice and in following the progress of cases of jaundice of any variety.

Urinary bilirubin and urobilinogen. For details see p. 83.

Bromsulphthaline (BSP) excretion

Bromsulphthaline excretion is a sensitive test of liver function in non-jaundiced patients. Less than 5% of an injected dose should remain in the serum 45 minutes after the intravenous injection of 5 mg/kg body weight.

Alkaline phosphatases

This group of isoenzymes originates chiefly in bone and in the liver. Serum levels are elevated in liver disease, particularly in biliary obstruction, intrahepatic or extrahepatic, and in secondary carcinoma (also of course in bone diseases, especially Paget's disease, which is not uncommon in elderly persons). The normal serum concentration in adults is from 4 to 13 King Armstrong units/100 ml (25–90 I.U./ml) and in children from 10 to 20 King Armstrong units/100 ml (70–150 I.U./ml).

Serum aminotransferases (transaminases)

These enzymes are present in all cells, but the concentration is greatest in the liver and in striated muscle. The serum concentrations therefore rise after any tissue damage (including cardiac infarction) but are par-

ticularly increased in hepatocellular diseases, such as acute hepatitis. Varying figures for normal ranges are quoted, apparently depending on the temperature at which determinations are made. For practical purposes it may be taken that the normal level of both aspartate aminotransferase (AspT:GOT) and of alanine aminotransferase (AlaT:GPT) are less than 18 i.u./litre (40 spectrophotometric units/ml) of serum. In obstructive jaundice the level is usually moderately raised, but in severe hepatocellular disease very high levels (e.g. 1000 i.u./litre or 2000 spectrophotometric units/ml) may be found.

Plasma proteins

Albumin is produced by liver cells and plasma levels are lowered in hepatocellular disease, roughly in proportion to the severity of the illness. In severe liver disease the normal level of 3.6–4.7 g/100 ml may fall to 2 g or below. The liver has only a moderate reserve capacity for albumin synthesis, and so lowered levels may also be found if there is excessive loss of albumin through the kidneys, into the gut or from burns. Moderate falls in plasma albumin may also be found in any severe systemic disease.

The distribution of the main fractions of the plasma proteins—albumin, α_1 - and α_2 -globulins, β -globulin and γ -globulin—can be determined by various forms of electrophoresis and may show a characteristic pattern in hepatitis, as well as in other diseases such as the nephrotic syndrome, multiple myelomatosis and hypogammaglobulinaemia. For a discussion of this subject larger works must be consulted.

Plasma prothrombin

Since prothrombin is formed in the liver from vitamin K, the prothrombin index (p. 147) is reduced in obstructive jaundice, when vitamin K is not absorbed, and in advanced liver disease when prothrombin is not formed in the liver. A low prothrombin index, persisting after injections of synthetic vitamin K (menaphthone), suggests severe impairment of liver functions.

Liver function tests are employed mainly:

1. *To assist in the differential diagnosis between jaundice due to hepatitis and that due to biliary obstruction.* The diagnosis is made mainly upon consideration of the history and physical signs, but complementary tests are valuable. Absence of urobilinogen from the urine (p. 83) indicates complete obstruction. This rarely persists for long in hepatitis and is usually intermittent in gall stones. Thus persistent complete obstruction, demonstrated by repeated negative urinary urobilinogen tests, favour obstructive jaundice due to growth.

In jaundice due to biliary obstruction, liver cell function is normal at first and fails gradually. In acute hepatitis, liver cell dysfunction is maximum at first and usually lessens. Thus, in chronic jaundice repeated liver function tests showing deterioration in function suggest obstructive jaundice, while tests showing improvement of function suggest hepatitis.

The alkaline phosphatase is raised early in obstructive jaundice, but not in hepatitis. Thus, alkaline phosphatase levels above 40 units with normal or near normal transaminase levels suggest obstruction, while phosphatase levels below 15 with raised transaminase levels suggest hepatitis. Chlorpromazine and other drugs, however, may produce jaundice with liver function tests identical with those of biliary obstruction. (The finding of hepatitis B antigen—Australia antigen or HBAg—is strong evidence of hepatitis, though carriers do occur.)

2. *To detect liver failure, either to confirm a clinical diagnosis of hepatic disease or to estimate prognosis when operation is contemplated.* For this purpose several tests should be performed. In advanced cirrhosis and in hepatic failure from other causes, typical findings are a low plasma albumen (less than 3 g/100 ml), a low prothombin index, raised plasma bilirubin, a raised alkaline phosphatase level and a diminished excretion of BSP. The alkaline phosphatase may be raised, in the absence of jaundice, in primary carcinoma of the pancreas, in the presence of hepatic secondaries from any source and in bone disease. Reliance must not be placed exclusively upon the results of liver function tests, which are often unhelpful and occasionally misleading.

Liver scans

Liver scans with the aid of a radioactive material, usually colloidal gold, are used to show the size of the liver and to demonstrate lesions which do not take up the isotope, such as primary or secondary tumours, abscesses and hydatid cysts. These appear as filling defects.

Needle biopsy

Needling the liver was for many years the standard method for the diagnosis of liver abscesses (p. 53). In the last few decades it has been used to obtain material for histological study. There is a tiny but definite mortality from this procedure. It should not be regarded as a routine diagnostic method and its use should be restricted to special centres and those well trained in the technique employed.

EXAMINATION OF VOMIT

The character of the vomit varies with the nature of the food ingested and the absence or presence of bile. In *pyloric stenosis* the vomit is apt to be copious and sour-smelling, and exhibits a froth on the surface after standing. The presence of much *mucus* gives the vomit a viscid consistency. The appearance of the vomit in *haematemesis* varies in relation to the site and severity of the bleeding. If the bleeding is copious, the vomit may present the appearance of pure blood and contain clots. Such bleeding may come from a gastric ulcer or from the oesophageal varices of portal obstruction. More commonly the blood is altered in colour by being in contact with the gastric juice; it may be blackish in colour or dark brown. The latter appearance is due to the conversion of haemoglobin into haematin. The altered blood gives to the vomit an appearance often compared to that of *coffee-grounds*. The taking of preparations of iron or red wines may produce a similar appearance in the vomit. Vomit which contains dark-green bile may resemble vomit which contains blood. On diluting with water, however, the green colour of the bile becomes more apparent, while blood remains dark. If there is any doubt, the tests for blood described under the examination of faeces may be applied. Remember that blood in vomit may have come from the nose or lungs and have been swallowed. Faecal vomit, characteristic of advanced *intestinal obstruction*, is brownish black in colour and may resemble altered blood, but has a typically faecal odour.

EXAMINATION OF FAECES

Examination of the faeces is an investigation of great importance all too frequently omitted. No patient with bowel disturbance has been properly examined until the stools have been inspected. The white surface of a bedpan makes an ideal background for the detection of blood, pus and mucus.

Naked-eye inspection

The following points should be noted:

The *amount*. It is sufficient to state whether the stools are copious or scanty.

The *colour*. *Black* stools may be produced by the ingestion of iron or bismuth. In haemorrhage occurring high up in the intestine the altered blood makes the stools dark, tarry-looking and very offensive, and all chemical tests for blood are strongly positive. *Pallor* of the stools may be due to lack of entrance of bile into the intestine, as in obstructive

jaundice; to dilution and rapid passage of the stool through the intestine as in diarrhoea; or to an abnormally high fat content as in malabsorption.

The *odour*. The stools in jaundice are often very offensive. Cholera stools, on the other hand, contain very little organic matter, and are almost free from odour. The stools of acute bacillary dysentery are almost odourless, while those of amoebic dysentery have a characteristic odour, something like that of semen.

The *form* and *consistency*. In constipation they may be drier and harder than normal, and sometimes resemble sheep's stools. In all forms of diarrhoea they are more fluid than normal, and may be watery. Slimy stools are due to presence of an excess of mucus.

To detect *abnormal ingredients*, the stool should be placed on a fine sieve, and a large quantity of water added. The whole is shaken and stirred up till the soluble parts are all washed away. The residue is then examined. The head of a tapeworm can best be seen if this residue is strained through black muslin. The head is about as large as that of a large pin, and the neck about as thick as a stout thread.

Watery stools are found in all cases of profuse diarrhoea, and after the administration of purgatives. To the stools of cholera the special name of *rice-water* stools is applied. Such a stool is colourless, almost devoid of odour, alkaline in reaction and contains a number of small flocculi consisting of shreds of epithelium and particles of mucus. Purulent or pus-containing stools are found in severe dysentery or ulcerative colitis, or in cases where an abscess has found its way into the intestines. Slimy stools are due to the presence of an excess of mucus, and point to an affection of the large bowel. The mucus may envelop the faecal masses, or may be intimately mixed with them. Bloody stools vary in appearance according to the site of the haemorrhage. If the bleeding takes place high up, the stools look like tar. In an intussusception they may look like red-currant jelly. If the haemorrhage is from the large intestine, the blood is less intimately mixed with the faecal matter, and may even be of a bright colour. In haemorrhage from the rectum or anus it may merely streak the faecal masses. The stools of bacillary dysentery consist at first of faecal material mixed with blood and pus, later of blood and pus without faecal material. Those of amoebic dysentery characteristically consist of fluid faecal material, mucus and small amounts of blood. The stools of steatorrhoea are very large, pale and putty-like or porridge-like and sometimes frothy. They are apt to stick to the sides of the lavatory pan and are difficult to flush away. If formed, they usually float.

Chemical examination

Tests for occult haemorrhage

Since benzidine ceased to be available, on account of a high incidence of carcinoma of the bladder in those who made it, orthotolidine has taken its place.

Orthotolidine* test. A portion of faeces the size of a pea is suspended in 5 ml of distilled water and boiled for 5 minutes. A 4% stock solution of orthotolidine in 95% ethyl alcohol is prepared. For use a 1 in 5 solution is made in glacial acetic acid. 1 ml of this reagent, 0.25 ml of the faecal suspension and 0.25 ml of hydrogen peroxide (20 vol. strength) are mixed in a test tube, and the result read in 3 minutes. A strong positive is indicated by a dark green colour and a weak positive by a pale green. This is a moderately sensitive test, and to avoid false positives the patient should have been on a meat-free diet for 3 days before the test and should not have been taking aspirin.

Haematest* tablet test. These tablets contain orthotolidine and strontium peroxide. A blue colour is produced when a wetted tablet is in contact with faeces containing blood.

A thin smear of faeces is made on the test paper provided. A tablet is placed in the centre of the smear, and two drops of water are run on to the tablet. The water overflows on to the smear of faeces. A slight blue colour on the tablet is of no significance, but a blue colour developing in the smear at the edge of the tablet within 2 minutes is a positive result.

This test is relatively insensitive. It can therefore be used on patients on a normal diet, but will not detect small amounts of gastrointestinal bleeding.

Both tests are of value in indicating the presence of gastrointestinal bleeding, but both may be negative in the presence of lesions which bleed intermittently or slightly, particular those situated in the upper gastrointestinal tract. Spectroscopic methods and isotopic methods using radioactive chromium labelled red cells are also available.

Haemastix. As described under urine (p. 78), this can be used as a rough screening test for blood in faeces.

Fats in faeces

Fat is present in food as neutral fat or triglyceride. It is split to greater or lesser degree by lipases, mainly of the pancreas, into glycerol and fatty acids. Some of the fatty acids, if unabsorbed, combine with bases to

*Not available in the UK (1974).

form soaps. Fat may, therefore, be found in the faeces as neutral fat, fatty acids and soaps.

The estimation of the proportion of split and unsplit fats present has been found unreliable as a method of distinguishing pancreatic from non-pancreatic steatorrhoea because of the effects of bacterial activity on neutral fats.

For the estimation of the fat in the stools, the patient may be placed on a diet containing 50 g of fat per day. The fat present in the stools collected over three or better five days is then estimated and should not exceed 6 g/day (or 12 g in 3 days). It has been found that equally reliable results are obtained if the patient eats a normal diet provided a 3 to 5 day collection is made.

Microscopical examination

See protozoa in faeces (p. 68).

INTESTINAL PARASITES

The parasites which occur in the intestinal tract include worms and protozoa. Some of the nematode and cestode worms will be described.

Nematoda

The commonest of all internal parasites is the threadworm, *Enterobius vermicularis*, whose presence is associated with considerable itching about the anus. It inhabits the large intestines, caecum and appendix, and female specimens can often be seen wriggling about in the recently passed motion of their host. To the naked eye they look like small white threads, 0.5 to 1 cm in length. Under the microscope the female may be distinguished by her much larger size, by the large uterus filled with ova, and the pointed posterior end. For appearance of ovum, see Plate VIII.

Ascaris lumbricoides, the roundworm, has a general resemblance to an earthworm. It usually measures up to 25 cm but may be up to 33 cm long. The ova, which can occasionally be found in the faeces, have brownish-yellow granular contents, and in many cases the shell is surrounded by an irregular sheath (Plate VIII).

Ankylostoma duodenale, the hookworm, is an important cause of anaemia and debility in the tropics, where heavy infestations may occur. It is no longer indigenous in Great Britain, but may be found in immigrants. It lives for the most part in the upper part of the jejunum, and its presence there is probable when, in an infested district, severe anaemia,

otherwise inexplicable, sets in. The diagnosis is confirmed by the discovery of ova in the motions (Plate VIII). They exhibit a segmented yolk enclosed in a thin shell, and are sufficiently numerous to be readily detected. The adult worm, which is rarely seen before therapeutic agents have been employed, is about 1 cm long, and the mouth is provided with four claw-like teeth.

Trichinella spiralis gains access to the body as the result of the eating of infested pork. Trichiniasis is rare when pork is eaten cooked, but small outbreaks and sporadic cases have been reported in Great Britain and in the USA. When man ingests the muscle trichinellae of the pig, larvae are set free in the small intestine, giving rise to the symptoms of the first stage of the illness—abdominal pain, vomiting and diarrhoea. The adult female, 3 mm long, penetrates the intestinal wall and discharges embryos into lymph spaces, whence they migrate into muscles. In this second stage of the illness the patient has fever and high eosinophilia and the muscles swell and become hard and tender. Rarely death may occur at the height of the myositis. Otherwise the embryo undergoes no further development, and its capsule becomes calcified. Unlike cysticerci, described below, calcified trichinellae are not visible in X-rays.

Cestoda

Many different kinds of tapeworm have been found as parasites in man, but the most important are *Taenia saginata*, *T. solium* and *Echinococcus granulosus*. Besides its occurrence in the fully developed state, *T. solium* may be present in the tissues in the form of a cysticercus; *T. saginata* is never found in this condition in man; whilst *E. granulosus* always occurs in the cystic stage, and has never been found in the mature condition in the human intestinal tract.

The presence of an adult tapeworm in the bowel is generally revealed by the passage of ripe proglottides in the stools, and after the administration of anthelmintics, the head may be detected by the method previously described (p. 63).

Taenia saginata (*mediocanellata*) is the beef tapeworm. Infestation occurs as a result of consuming insufficiently cooked beef infested with the embryo of the worm. The adult parasite reaches a length of 4 to 8 m and consists of about 2000 segments. The ripe proglottides measure 13 by 6 mm. The head is quadrate, measures 2 mm in diameter, has 4 suckers, but is devoid of hooklets. The terminal gravid segments of the worm become separated from time to time and the ova may be then ingested by the bullock or cow, in the muscles of which the larva develops. It becomes a bladder worm, *Cysticercus bovis*, measuring 10 by 6 mm and containing an invaginated head which possesses in miniature the characteristics of the adult scolex. *Cysticercus bovis* is

never found in human muscle tissue or brain. For appearance of ovum, see Plate VIII.

Taenia solium, the pork tapeworm, is not encountered in Britain but is endemic wherever infested pork is eaten raw or insufficiently cooked. It measures 2 to 4 m in length; a ripe proglottis is 10 by 5 mm. The head measures 1 mm in diameter and in addition to 4 suckers has a rostellum with 32 hooklets. The ova in the terminal proglottides may be ingested by the pig, in the muscles of which the bladder worm *Cysticercus cellulosae* develops. Occasionally persons in infected areas become infested with *Cysticercus cellulosae* from eating food contaminated with the ova of the parasite. The muscles of the human host are then infested by cysticerci, which are palpable through the skin as tense ovoid swellings 10 by 5 mm and of almost cartilaginous hardness. About 4 years after infestation they become calcified and may then be demonstrated radiologically. The thigh muscles are those in which cysticerci are most easily demonstrated. Cysticerci may also occur in the brain and are a cause of epilepsy.

Echinococcus granulosus. The adult worm, which consists of a head and three segments, and whose length is only 3 to 8 mm, need not be fully described, since it is not found in man. The *cystic stage* is very important, as it gives rise to serious disease in man in many of the viscera and especially in the liver. The cysts of this are not simple, but produce from their inner surface one or two generations of secondary vesicles, on which the brood capsules, containing the cestode heads, are formed. During the period in which this process is going on, the primary vesicle dilates to accommodate its increasing contents, and may eventually reach the size of a coconut. The vesicles may rupture spontaneously, and their contents may escape by the lungs, by the bowel or by the urinary passages. Specimens may be obtained by aspiration or after surgical interference.

The diagnosis of suspected hydatid disease may rest upon the recognition of the nature of fluid withdrawn, of hooklets or scolices, or the appearance of parts of the ectocyst, which are sometimes coughed up from the lungs. The Casoni intradermal test may be of some assistance, though cross reactions may occur in infestation with other cestodes.

The *fluid* is clear, alkaline, devoid of protein, and contains abundance of sodium chloride and traces of glucose. Its density is low, being generally under 1010. The *scolex*, if it is obtained in a perfect condition, is about 0.3 mm in diameter, and a number of them often spring in a group from one brood capsule. They have 4 suckers and a crown of hooklets. Portions of the *ectocyst* appear as whitish-yellow shreds, which can be recognized under the microscope by their lamination and by the pectinate markings on the laminae.

Diphyllbothrium latum (*Dibothriocephalus latus*), the fish tapeworm,

is encountered in Sweden, Finland and Michigan. The adult worm measures from 3 to 10 m or more and has a total of 3000 segments. The scolex is small, spatula-shaped and possesses two deep suckorial grooves. The first larval host is a water flea and the second the pike, perch or salmon trout. Human infestation takes place from eating raw or undercooked fish. In a tiny proportion of cases, a vitamin B12 deficiency anaemia may be produced.

Trematodes

Schistosomes or blood flukes are the most important trematode parasites of man. They are found in three varieties, and produce the disease known as schistosomiasis or bilharziasis. *Schistosoma japonicum* and *Schistosoma mansoni* inhabit the portal blood stream, and the ova (Plate VIII) are passed in the faeces. *Schistosoma haematobium* characteristically inhabits the vesical plexus, so that the ova (Plate VIII) are passed in the urine (p. 86). They may occasionally also be found in the faeces. *Schistosoma mansoni* is found in Africa and South America; *Schistosoma haematobium* in Africa and the Near East, particularly in Egypt; and *Schistosoma japonicum* in the Orient.

Protozoa

A number of protozoa, many of them non-pathogenic, have been found in the faeces. Of these the most important clinically is *Entamoeba histolytica* (Plate VIII), which causes amoebic dysentery (as opposed to bacillary dysentery) and sometimes tropical abscess of the liver. *Entamoeba coli* (Plate VIII) is non-pathogenic.

Entamoeba histolytica is found in the stools in two forms. In acute attacks vegetative amoebae can generally be found, whilst in the more quiescent stage cysts are passed. So-called 'small' and 'large' races of *Entamoeba histolytica* have been described. It is likely that both are pathogenic.

If amoebic dysentery is suspected, a stool should be passed into a clean bedpan; this must be free from antiseptics, and the stool must not be mixed with urine. It should be taken immediately to the laboratory so that it is examined whilst warm.

With a platinum loop, select a piece of blood-stained mucus, or failing this, a small particle of faeces; emulsify it with a drop of warmed (37°C) normal saline and apply a cover-slip.

The diagnosis of vegetative *Entamoeba histolytica* depends for practical purposes on the demonstration of actively motile amoebae, which contain red cells. The slide must be examined on a heated stage, or, if this is not available, it may be kept warm by applying coins pre-heated in a Bunsen flame, on each side of the cover-slip. Care must be taken not to overheat it. Motile amoebae

are readily seen under the low power, and can be studied further under the 16 mm objective. Iodine will kill the amoebae, and must not be used.

Cysts can be seen under the low power as small round refractile bodies. They are seen even better if the stool is emulsified in 1% aqueous eosin, when, provided the stool is fresh, they show as white bodies against a pink background. The characteristic chromatoid bodies of *E. histolytica* are well shown by this method. Globules of oil or fat, which may be present in the faeces of

TABLE 3. *ENTAMOEBA HISTOLYTICA* AND *ENTAMOEBA COLI*: VEGETATIVE FORMS

	<i>Entamoeba histolytica</i>	<i>Entamoeba coli</i>
Occurrence	Fairly abundant, when present in amoebic dysenteric stools	Never abundant. Occasionally seen in dysenteric stools
Size	Variable, average 20 to 30 μ m	Less variable. Generally larger than <i>E. histolytica</i>
Motility	Active. Large pseudopodia. These become larger as activity diminishes before death	Sluggish. Small cone-shaped pseudopodia
Cytoplasm	Homogeneous and 'ground-glass like'. Differentiation of ectoplasm and endoplasm clearly seen. Red blood corpuscles often seen	Appearance porcellanous. Ectoplasm less plentiful, line of demarcation between it and endoplasm inconspicuous. Endoplasm granular. Abundant food vacuoles with usually bacterial inclusions. Red blood corpuscles never present
Nucleus	Karyosome, not seen in unstained preparations, small and <i>central</i> . Periphery marked by ring-like layer of regular sized chromatin granules	Distinct. Karyosome usually large, irregular and <i>nearly always eccentric</i> . Ring-like layers of peripheral granules more pronounced and irregular in size and shape

patients who have been given oil as an aperient, may resemble them, and, if numerous, may make any attempt at further examination useless. Oil droplets vary in size, are structureless, and their edges cannot be sharply focused. If cysts are present, iodine should be used for their further identification. Make a further preparation using 1% Lugol's iodine. Find a suspected cyst under the low power. Apply the 16 mm objective and centre the cyst in the middle of the field. Rack up the microscope tube, apply a small drop of oil to the cover-slip without moving it, and carefully lower the oil-immersion objective into the drop. The main differences between *E. histolytica* and *E. coli* and their cysts are shown in Tables 3 and 4.

TABLE 4. *ENTAMOEBA HISTOLYTICA* AND *ENTAMOEBA COLI*: CYSTS

<i>Entamoeba histolytica</i>	<i>Entamoeba coli</i>
When mature, 4 nuclei, with nuclear karyosomes central. Peripheral chromatin often semilunar	When mature, 8 nuclei, with nuclear karyosomes usually eccentric
Size slightly smaller on average, varying from 10 to 20 μm	Size slightly larger, varying from 10 to 20 μm
Glycogen less abundant	Glycogen more abundant
Refractility moderate	Refractility considerable
Rod-shaped 'chromidial bodies' usually seen in fresh specimen	'Chromatoid bodies' not often present: thread-like or in bundles when seen
Cyst wall rather thinner	Cyst wall rather thicker

Besides *E. coli*, there are three other non-pathogenic amoebae, which must not be mistaken for *E. histolytica*. They are *Endolimax nana*, *Iodamoeba bütschlii* and *Dientamoeba fragilis*. In the diagnosis of the vegetative forms this mistake will be avoided if it is remembered that *E. histolytica* alone is actively motile and contains red cells. The cysts of *Iodamoeba bütschlii* (Plate VIII) contain a single small nucleus, an eccentric karyosome and a very large compact mass of glycogen.

Giardia lamblia is a flagellate protozoon, which inhabits the duodenum and may be found in the stools of patients with diarrhoea in both cystic and

vegetative form (Plate VIII). There is some doubt whether or not it is pathogenic. The fact that a course of mepacrine will eradicate it, sometimes with relief of the diarrhoea, suggests that it is.

Trichomonas hominis (Plate VIII) is another flagellate protozoon, which may be seen in the stools in diarrhoea. It is probably non-pathogenic. Similar if not identical trichomonas may be found in the vagina in leucorrhoea and in the mouth in oral sepsis.

Isospora belli (*I. hominis*) (Plate VIII), one of a group of parasites which may produce coccidiosis in man, animals and birds, has occasionally been described as the cause of acute diarrhoea in man, particularly in the eastern Mediterranean.

Balantidium coli (Plate VIII) is a large ciliate protozoon which is found in the intestine of pigs. It occasionally infects man, and may rarely cause severe diarrhoea or frank dysentery.

4

THE URINE

Physical Examination — Chemical Examination — Microscopical Examination — Bacteriological Examination — Estimation of Renal Efficiency — Other Investigations

Sir Robert Hutchison used to say that the ghosts of dead patients that haunt us at the midnight hour do not ask why we did not employ the latest fad of clinical investigation: they ask 'Why did you not test my urine?' or 'Why did you not put a finger in my rectum?'

To the risk that the urine may not be tested at all new dangers have been added, in that urine testing is increasingly delegated to non-medical persons, who may be inadequately trained in the performance and interpretation of the tests they use and unaware of their importance. All doctors and students should be thoroughly conversant with urine testing, which is an essential part of any medical examination.

Urine passed into a clean glass vessel is suitable for routine urine testing. Special methods designed to reduce urethral and other contamination are desirable for microscopical examination (p. 84) and essential for bacteriological examination (p. 86).

The urine should be examined (a) physically, (b) chemically, (c) microscopically and (d) bacteriologically.

PHYSICAL EXAMINATION

Attention should be paid to the following points: (a) quantity; (b) colour and transparency; (c) specific gravity; and (d) naked-eye characters of the deposit.

Quantity

The normal quantity of urine passed daily varies widely—from 700 to 2500 ml—depending on the fluid intake. Normally, very much more urine is secreted during the day than during the night. Approximation of the night quantity to that of the day is always abnormal, and occurs especially in renal failure, of which it may constitute one of the earliest signs. Thus, *nocturnal polyuria*—a symptom which is now often referred to by the unfortunate term 'nocturia'—may be the first symptom of

failure of the concentrating power of the kidney and may indicate the onset of renal failure.

An *increased secretion* of urine occurs physiologically after increased consumption of food or drink, and after exposure to cold. Conversely, *diminished secretion* occurs when little food or drink has been taken, and after heat-induced sweating.

A *pathological increase* is an early sign of chronic renal failure. Polyuria is also a feature of diabetes mellitus and diabetes insipidus, and accompanies the elimination of oedema fluid. Abnormal reduction of urine output may be due to salt and water depletion from diarrhoea, vomiting or fever; the sudden lowering of blood pressure; severe heart failure; or acute diffuse disease of the kidney tissue such as occurs in post-streptococcal glomerulonephritis.

Complete cessation of urine output (*anuria*) is uncommon and most often results from obstruction.

Colour and transparency

Normal urine has the colour of amber or pale sherry. The exact tint fluctuates widely. The concentration of the urine cannot be estimated from its appearance.

Small quantities of blood give the urine a smoky appearance and larger quantities make it brownish or red. Haemoglobin in large quantities, as in blackwater fever, gives it a colour varying from dark red (port wine) to brownish black or even almost black. The presence of bile gives it a brown or even dark brown colour. The urine is abnormally pale when it is very dilute, i.e. with specific gravity of 1002 or 1003, and in renal failure, when the normal colouring matter (urochrome) is greatly diminished or absent.

The taking of drugs may also lead to discoloration of the urine. Some examples are anthracene purgatives (orange coloured); desferrioxamine (reddish brown); phenylindanedione (pink); furazolidone and niridazole (brown); rifampicin and phenazopyridium (red); methylene blue, present in some proprietary kidney pills (green); and methyldopa and iron sorbitol (grey or black).

Normally, when freshly passed, urine is quite transparent, but it may be *opalescent* from the presence of various substances in suspension, of which the most important are pus, bacteria and phosphates. Phosphates disappear on adding acid. If the opalescence persists after filtration, it is due to the presence of bacteria. Urine that has cooled may be cloudy from the presence of urates. In this case it will become clear again when warmed.

Specific gravity

The concentration of urine is best expressed as its *osmolality*, which depends on the number of osmotically active solute particles per unit of solvent, and this may be determined by measuring its freezing point.

Although less accurate it is often helpful, and usually more practical, to measure the *specific gravity*, which depends on the type as well as the number of solute particles, with the aid of a *urinometer*. In order to obtain satisfactory results certain precautions must be taken. The glassware used should be clean and the manometer should float freely in the measuring cylinder. The urinometer reading at the bottom of the meniscus is the relevant one. As the urinometer is calibrated at 15°C, a falsely low reading will be obtained if urine is tested whilst warm shortly after being passed, so it should be allowed to cool to room temperature. If there is insufficient urine for the manometer to float freely, the urine should be diluted with an equal volume of distilled water and the last two figures of the urinometer reading doubled. Randomly measured, the normal specific gravity varies from 1001 to 1025, depending on the state of hydration and the time of day. It may occasionally rise to 1035 even in health.

In normal urine the specific gravity is proportional to the urinary concentration of urea and sodium. An abundant urine of high specific gravity is found if the urine contains many heavy particles, e.g. radiographic contrast media, glucose (as in diabetes mellitus) or protein (1 % of protein increases the specific gravity by 3 points). In diabetes insipidus, on the other hand, the specific gravity may fall almost to that of distilled water, and this may also happen in hysterical polydipsia.

Serial examinations of the specific gravity constitute the simplest and one of the most valuable methods of observing renal function. With normal kidneys the concentration of the urine varies to a considerable extent. As renal failure develops the specific gravity approximates more closely to 1010 the specific gravity of the glomerular filtrate. With complete loss of concentrating power the specific gravity becomes fixed at 1010, the urine then being isotonic with the plasma water. The specimen passed on rising should always be tested for this purpose, as it is normally the most concentrated that is passed in the day. In the normal person with a normal protein intake the specific gravity should be at least 1020, if no fluid has been drunk for 9 hours. A longer period of fluid deprivation is required to achieve maximum urine concentration, but care must be taken that the patient does not become too dehydrated.

Naked-eye characters of the deposit

Normal urine is perfectly clear and transparent when voided. After it has stood for some time a deposit of 'mucus' appears in it. This forms a

woolly-looking cloud, which usually settles to the bottom of the glass, but if the urine is of high specific gravity, it may be in the middle of the glass, or even at the top. If traces of blood are present in the urine, the cloud of 'mucus' may have a brownish tint.

The normal urinary ingredients, which may separate out in the form of a deposit visible to the naked eye, are phosphates, urates and uric acid. Phosphates separate out as a white deposit when the urine is neutral or alkaline and especially when it is heated. The deposit is dissolved by acetic acid. Urates and uric acid form yellow, red or brown deposits, particularly if the urine is concentrated or highly acid; they disappear on warming. Although usually of no significance, they may appear when purine breakdown is increased, as in myeloproliferative disorders, especially after treatment, and may cause renal colic or even urinary obstruction.

CHEMICAL EXAMINATION

Many of the traditional chemical tests for urinary constituents have been replaced for routine purposes by commercial tablet or reagent stick tests. Some of these will be described briefly, but for full details the manufacturer's literature should be consulted. *Failure to follow the manufacturer's instructions exactly, or failure to keep the reagents in a satisfactory condition, will result in incorrect deductions;* the indicator ends of reagent strips should not be handled.

pH

It is customary to test the reaction of the urine with pH indicator papers, but the result is rarely important except when a drug has been given with the intention of altering the pH for therapeutic purposes. Normal urine is nearly always acid. Rarely it is repeatedly neutral or alkaline, when the patient is not taking alkalis, and this may indicate impairment of the power of the tubules to excrete acid. This can be confirmed by accurate measurements of the pH of the urine after the patient has taken ammonium chloride (p. 89).

Proteins

Before proceeding to apply the 'traditional' tests for protein it is essential that the urine should be absolutely clear. It may therefore be necessary to filter it. If, after filtering more than once, the urine remains turbid, bacteria are probably present, and can be removed by long centrifugation or by shaking up the urine with powdered barium carbonate and filtering. If the turbidity is due to urates, it will disappear when the urine is heated; if due to phosphates it will disappear after acidification.

Commercial reagent strips

These strips (e.g. Albustix) are impregnated with buffered tetrabromophenol blue, and change from yellow to various shades of green depending on the amount of protein in the urine. The colour of the active part of the strip is compared with the colours on the maker's chart immediately after the stick has been transiently dipped in the urine. The test is most sensitive for albumin, and the amount of albumin present in normal urine (about 30 mg/100 ml) may be indicated on the stick as a 'trace'. Fluorescent light may make appreciation of the colour change difficult. False positive results may occur if the urine is very alkaline, or during treatment with phenothiazines. The test may give a negative result with urine containing Bence Jones protein.

Sulphosalicylic acid test

This test for protein is very reliable, is semiquantitative, and does not require heat. In a test tube place about 5 ml of urine. Filter if cloudy, add 6 drops of 20% sulphosalicylic acid. The formation of a cloudy precipitate indicates the presence of protein. The cloud is seen best when looked for against a black background. For ordinary purposes it is often sufficient to express the amount present as a haze, a cloud or a granular deposit. A heavier deposit may be allowed to settle and the quantity may be expressed as the proportion of the total urine volume occupied by the deposit. If this proportion is one half, the urine contains about 1 g protein/100 ml. A 'cloud' of protein represents only about 20 mg/100 ml. False positives may be due to turbidity in the urine (in which event the specimen should be filtered) or the presence of radiographic contrast media; they may also occur with the urine of patients treated with sulphonamides, tolbutamide, para-aminosalicylic acid or large doses of penicillin, or if the urine contains a lot of uric acid.

Boiling test

This is a satisfactory test for protein, but needs to be carried out with care.

Fill a small test tube two-thirds full of urine. If the urine is alkaline, add a small piece of litmus paper to the urine and then add 10% acetic acid, drop by drop, mixing thoroughly after each drop, until the litmus paper is just red. Incline at an angle, boil the top 2 cm over a flame holding the bottom of the tube, and examine against a dark background. A cloudiness indicates the presence of either protein or phosphates. Add 10% acetic acid drop by drop, and boil. If the cloud disappears, it consists of phosphates; if it persists, protein is present. Acid should be added drop by drop till no further precipitation of protein occurs. If there is more than a light cloud, add a few more drops of 10% acetic

acid, mix well, and, holding the test tube in a holder, boil the whole volume. The test tube is put aside for half an hour or more for the protein to settle when it can be expressed semi-quantitatively as described for the sulphosalicylic acid test. The test is comparable in sensitivity to the latter test, but is not very reliable if the urine is dilute. Treatment with tolbutamide, large doses of penicillin or the presence of radiographic contrast media may result in false positive results.

If proteinuria is detected by either the sulphosalicylic acid or boiling test, but not by Albustix, the presence of Bence Jones proteinuria should be suspected. This is confirmed if a precipitate forms as the urine is heated, only to disappear on boiling and re-precipitate during cooling; however, more sophisticated laboratory tests may be needed to detect it.

Quantitative estimation of protein

This can be done with sufficient accuracy for clinical purposes by means of Esbach's albuminometer. The principle of the method consists in measuring the depth of the coagulum produced in the urine by the addition of picric acid. The instrument consists of a thick glass test tube, with graduations on it from 0 up to 7. To make up Esbach's reagent, dissolve 10 g of picric acid to 20 g of citric acid in about 900 ml of boiling water. Allow this to cool and add water to 1 litre. Filter the urine if not already clear, and if alkaline render slightly acid with acetic acid. If the specific gravity be 1010 or more, dilute the urine sufficiently to bring the density below that level (to 1008). This is important, and is often overlooked. Fill the tube with the urine up to the mark U. Pour in the reagent up to the mark R. Close the tube with a rubber stopper, and gently invert it a few times to allow the fluids to mix. Set aside for 24 hours. At the end of that time read off the level of the surface precipitate. If after the first trial the level of the precipitate is found to be above the mark 4, the urine must be diluted and a fresh estimation made. The figures on the scale represent grams of dried protein per litre of urine. Divide by 10 to get the percentage. If the urine requires to be diluted, the result must, of course, be multiplied the requisite number of times. This method yields only approximate results, since the precipitates obtained in different urines vary in compactness and in the length of time they take to settle. One should also remember that it measures only the concentration of protein in the urine and not the total amount. Very small quantities of protein cannot be estimated by Esbach's method, as the instrument does not record less than 0.1 %.

All these methods measure protein concentration and if urine output is abnormal this may give a misleading impression of the urinary protein excretion per 24 hours.

Normal urine contains a small quantity of protein—usually less than 20 mg/100 ml. Two-thirds of the protein consists of globulins; some of

these are identical with plasma globulins, whilst others come from tubular secretion or are added in prostatic and urethral secretion, and semen. The quantity excreted rarely produces more than a 'trace' reaction with tetrabromophenol blue reagent sticks, which are more sensitive to albumin. Rather more protein may be found in patients without renal disease during fever and in healthy persons after exercise. protein may be found in specimens collected randomly during the day, but not in the first urine specimen passed after arising (orthostatic proteinuria). Such proteinuria is usually of no importance. Persistent proteinuria usually indicates the presence of renal disease, but the amount does not indicate the severity of the disease. It may also be due to extrarenal disease, such as congestive heart failure. Diseases affecting the lower urinary tract usually cause only slight proteinuria.

Blood and its derivatives in urine

Whole blood may appear in the urine (haematuria), or blood pigment may appear without corpuscles (haemoglobinuria). These two conditions can only be differentiated by microscopical examination of the deposit for blood cells. This is particularly important for the detection of small numbers of red cells such as may be found for several days after an attack of renal colic and in bacterial endocarditis.

If urine contains only a small amount of blood or blood pigment it has a peculiar opaque appearance, to which the term 'smoky' is applied. Large quantities of blood give to the urine a red colour varying in intensity with the amount of blood present. The blood corpuscles are apt to settle at the bottom, producing a flocculent deposit, which is brown or red according to the amount of blood and the degree of its alteration.

The presence of blood may be confirmed by the finding of red cells under the microscope, or by a chemical test for haemoglobin such as Haemastix.

Occultest.* One drop of urine is placed in the centre of the test paper provided and one Occultest tablet is placed on the resulting moist area. Two drops of water are run on to the tablet. If no blue colour appears in 2 minutes, the test is negative. The presence of blood in the urine is indicated by the appearance of a diffuse blue colour on the paper around the tablet within 2 minutes of the addition of the water. The colour of the tablet itself is irrelevant.

Haemastix reagent slips. A blue colour developing in the test area of the strip, 30 seconds after it is momentarily dipped in the urine indicates

* Not available in the UK.

that blood or haemoglobin is present. This test depends on the oxidation of orthotolidine to a blue pigment; therefore the presence of extraneous oxidizing agents such as hypochlorites will produce a false positive result, and the presence of reducing substances (e.g. in patients taking ascorbic acid) may produce a false negative one.

Urine which contains blood or haemoglobin also, of course, contains some protein, and it is often difficult to say whether the blood is sufficient to account for all the protein present, or whether proteinuria exists in addition. If human blood is added to normal urine in an amount sufficient to produce distinct smokiness, the quantity of protein amounts to merely a trace. Even when the quantity added is sufficient to render the urine distinctly red, the amount of protein is only about $\frac{1}{2}$ per 1000.

Sugars in the urine

Glucose is by far the most important of the sugars which may appear in the urine. In normal people it occurs in amounts too small to be detected by the usual methods employed, and if it is so detected its presence may be regarded as pathological. Glycosuria may be due to excessive blood glucose levels as in diabetes mellitus or to defective renal tubular re-absorption—'renal' glycosuria.

The presence of a *reducing substance* in the urine may be detected by means of Benedict's test or by the use of Clinitest tablets, Clinistix strips or Diastix strips. The last two are specific for glucose.

Benedict's test. To 5 ml of Benedict's reagent add 8 drops of the urine, boil for 2 minutes and allow to cool. If a reducing substance is present, a precipitate will appear, varying from a light green turbidity to a red precipitate. If the reduction is due to glucose, the test gives approximately quantitative results:

Light green turbidity	0.1–0.5 % sugar
Green precipitate	0.5–1.0 % sugar
Yellow precipitate	1.0–2.0 % sugar
Red precipitate	2.0 % sugar or over

Clinitest. This is a convenient modification of Benedict's test, in which the ingredients are present in a tablet and the necessary heat is provided by the interaction of sodium hydroxide and citric acid. Five drops of urine are placed in a test tube with the dropper provided. The dropper is rinsed and 10 drops of water are added. One Clinitest tablet is dropped into the tube and the resulting reaction observed. Effervescence occurs, followed by boiling. Fifteen seconds after the boiling has ceased, the tube should be shaken gently and the colour of the contents compared with the colour scale provided. When a reducing substance is present, the copper sulphate in the solution is converted to cuprous

oxide, causing the colour to change through green (0.5%) to orange (2%). If a transient orange 'flash' is seen during the effervescence it indicates that the urine contains at least 2 g reducing substance per 100 ml irrespective of the final colour.

The presence of glucose in the urine can be detected by enzyme tests:

Clinistix. This test is specific for glucose, but is less easy to quantitate than Clinitest. The reagent strip is dipped transiently in the urine; the colour of the test area is compared with the maker's colour chart exactly 10 seconds later. A change from red towards purple indicates that the urine contains an abnormal amount of glucose.

Clinistix have a test area impregnated with a mixture of glucose oxidase, peroxidase and a chromogen system. In the presence of glucose oxidase, glucose is oxidized by atmospheric oxygen to gluconic acid and hydrogen peroxide. The latter, in the presence of peroxidase, oxidizes the chromogen system to a shade of purple. This test is specific for glucose and highly sensitive, unless inhibited by a high concentration of ascorbic acid in the urine, such as may occur with the oral administration of ascorbic acid or the parenteral administration of some tetracycline preparations.

Diastix is a modification of the above in which the results are semi-quantitative. The colour should be read after exactly 30 seconds.

The reducing substances which may be found in urine include glucose, lactose, fructose, pentose, homogentisic acid. Treatment with drugs such as ascorbic acid or cephalosporins may also give rise to a positive result with Benedict's test or Clinitest.

Homogentisic acid is present in the urine in alkaptonuria. Lactosuria occurs in late pregnancy and during lactation. Pentosuria is usually due to a rare inborn error of metabolism but may follow the ingestion of certain fruits.

In practice much the commonest and most important reducing substance in the urine is glucose and if a reducing substance is present in the urine and the patient has indubitable symptoms of diabetes mellitus, further confirmatory tests are unnecessary. If no symptoms are present, it is often simplest to resort immediately to the glucose tolerance test.

The glucose tolerance test. The usual method is as follows. The patient, who has been allowed no food since the previous evening, has blood taken for a fasting blood glucose test and empties his bladder. He then drinks 50 g of glucose dissolved in 100 ml of water. Further specimens of blood are withdrawn and further samples of urine collected at the end of $\frac{3}{4}$ hour and 2 hours (or after $\frac{1}{2}$, 1, $1\frac{1}{2}$ and 2 hours).

Under normal conditions the fasting blood glucose is between 80 and 120 mg/100 ml. The blood glucose $\frac{3}{4}$ hour after the test dose is taken has risen to 140 to 170 mg/100 ml, and at the end of 2 hours it has fallen to its fasting level. The corresponding specimens of urine contain no glucose, since glucose does not pass into the urine in detectable quantities in normal persons till the blood reaches 180 mg/100 ml—the so-called 'renal threshold'.

In renal glycosuria or 'lowered renal threshold' the blood glucose curve is normal, but glucose is found in one or more of the corresponding specimens of urine.

This test is employed in the diagnosis of diabetes mellitus. For this purpose it is essential that the patient should have been eating a normal amount of carbohydrate during the previous week. Normal persons on a low carbohydrate diet may show abnormal blood glucose levels after a test dose of glucose, and hence be misdiagnosed as cases of mild diabetes, if this precaution is not observed. Cases of severe diabetes mellitus can generally be recognized by the history and examination of the urine, but if the glucose tolerance test is performed the fasting blood glucose is usually well above 120 mg/100 ml and may be 200 or 300 mg. After the test dose of glucose the blood glucose rises and does not return to normal levels at the end of 2 hours. The glucose tolerance test finds its greatest usefulness, however, in excluding diabetes mellitus in persons with symptomless glycosuria.

Ketone bodies in the urine

Aceto-acetic acid and acetone (as well as hydroxybutyric acid, which is not a ketone) may appear in the urine of patients with severe diabetes mellitus, and often after starvation or prolonged vomiting. The ketones may be detected in the urine using Rothera's nitroprusside test or one of its modifications such as Ketotest or Acetest. Severe degrees of ketonuria may produce a positive ferric chloride (Gerhardt) test.

Rothera's test. The urine must be fresh and unboiled, because aceto-acetic acid is easily decomposed. 10 ml of the urine are saturated with ammonium sulphate by adding an excess of the crystals; 3 drops of a strong freshly prepared solution of sodium nitroprusside and 2 ml of strong ammonia solution are then added. A deep permanganate colour is produced. This test is given both by acetone and aceto-acetic acid but no other substances that may occur in fresh urine.

If Rothera's test is negative, ketone bodies are absent.

Acetest. This is a modification of Rothera's test in tablet form. One drop of fresh urine is allowed to fall on an Acetest tablet on a clean white

surface. A purple discoloration of the tablet 30 seconds later indicates the presence of aceto-acetic acid or acetone, and the amount can be roughly estimated by comparison with the makers' colour chart.

Ketostix. A Ketostix reagent strip is dipped momentarily in the fresh urine. A mauve colour of the test end of the strip after 15 seconds constitutes a positive result. Keto-Diastix reagent strips combine a semi-quantitative test for glucose with the test for ketones.

Gerhardt's test. 10% ferric chloride solution is added drop by drop to 5 ml of urine in a test tube. A precipitate of ferric phosphate usually forms, but disappears again when more ferric chloride is added. The solution becomes brownish-red if aceto-acetic acid is present.

Aspirin and other salicylates, phenothiazines, phenol and some other drugs give a similar colour with ferric chloride. Prolonged boiling (before adding the ferric chloride) destroys aceto-acetic acid, but the other substances which give a colour with ferric chloride are unaffected. If, therefore, urine which has been subjected to prolonged boiling still gives the ferric chloride reaction, it may be inferred that the reaction was not due to aceto-acetic acid. Boiling after adding ferric chloride destroys the colour, whether this is due to aceto-acetic acid or to other substances.

A positive ferric chloride reaction is obtained only if aceto-acetic acid is present in considerable amount. If, therefore, the urine reacts to Rothera's test but not to ferric chloride, it may be inferred that only small quantities of acetone bodies are present. If both are positive, the patient has a ketosis of considerable severity, demanding urgent treatment.

Bile in the urine

An increase in the concentration of circulating conjugated bilirubin results in its excretion into the urine, for it is water-soluble. The pigment makes the urine brownish in colour, but this is not specific for bile. A crude test for its presence is to shake the urine in a test tube. A positive result is given by the formation of a stable yellow froth; the stability is due to the presence of bile salts, and the colour to bilirubin. Ictotest is more sensitive and reliable.

Ictotest. Five drops of urine are placed on a square of the test mat provided. One Ictotest tablet is placed in the centre of the moistened area. Two drops of water are placed on the tablet. If bilirubin is present, the mat around the tablet turns bluish-purple. A pink or red colour should be ignored, as should any discoloration of the tablet.

Urobilinogen and urobilin in urine

Bilirubin secreted by the liver into the bile is reduced by the bacteria of the intestine to urobilinogen and urobilin. Some of this is reabsorbed and circulates in the blood stream. Of this a small amount is excreted in the urine, but the majority under normal circumstances is re-excreted by the liver into the bile. Estimation of the urobilinogen and urobilin in the urine may therefore be useful in several circumstances. In obstructive jaundice, a complete absence of urobilinogen and urobilin indicates that the obstruction is complete and that no bile pigment is reaching the intestine. In patients without jaundice an excess of urobilinogen and urobilin in the urine is due to the inability of the liver to excrete these substances into the bile and indicates hepatic dysfunction. Such an excess is often present in the pre-icteric stage of infective hepatitis and in diffuse diseases of the liver, such as severe cirrhosis. But the most important cause of an increase of urobilin and urobilinogen in the urine is excessive haemolysis. Although the haemolysis may be sufficiently marked to cause hyperbilirubinaemia, the bilirubin is largely unconjugated, is not water-soluble and is not excreted in the urine. An excess of urobilinogen in the urine can be detected and measured semi-quantitatively with Urobilistix reagent slips (see makers' instructions). They are valuable in the screening of persons exposed to hepatitis virus and to hepatotoxic substances.

The following chemical test has the advantage that it can detect porphobilinogen as well as urobilinogen.

Urobilinogen, which is colourless, condenses with Ehrlich's aldehyde reagent in acid solution to form red dyes which can be extracted by a mixture of amyl and benzyl alcohols. The test must be performed on urine which has been recently passed, for if it is allowed to stand urobilinogen undergoes spontaneous oxidation to urobilin, which does not give a positive result. Porphobilinogen (which is excreted in the urine in certain types of porphyria and condenses to porphyrins, which give the urine a port wine colour if allowed to stand) also produces a red colour in the test, but the colour is not extracted into the alcohol phase.

One ml of fresh urine at room temperature is mixed with 1 ml of Ehrlich's aldehyde reagent (2 g paradimethylaminobenzaldehyde, dissolved in 100 ml of 5% hydrochloric acid). After 1½ minutes 2 ml saturated aqueous sodium acetate are added and mixed, followed by 2 ml of a 3:1 (v/v) mixture of amyl alcohol and benzyl alcohol. The test tube is stoppered and its contents are shaken gently for 1 minute. After the phases have separated a red colour of the upper (organic) phase indicates that urobilinogen is present, whilst a similar colour in the lower (aqueous) phase denotes porphobilinogen. Fresh urine normally contains a little urobilinogen, which may be sufficient to produce a slight pink discoloration in the test. Urine diluted ten times with water will not do so.

MICROSCOPICAL EXAMINATION

It is customary to microscope the deposit of urine after it has been centrifuged at 1000–1500 rpm for about 3 minutes and placed under a cover slip. This is useful for quantitative examination, but it should be remembered that fragile items, such as casts, may be disrupted by prolonged or rapid centrifugation. Cells and casts disintegrate rapidly if the urine is allowed to stand, and it is essential to examine fresh urine if red, white and epithelial cells are to be distinguished. Unstained cellular elements, and particularly casts, may not be very refractile. They are best seen if the microscope diaphragm is partially closed and the condenser racked down.

Red blood corpuscles (Plate V)

Typically erythrocytes appear as roughly circular elements of about $7\text{ }\mu\text{m}$ diameter with a clear yellowish centre. If the urine is concentrated they are shrunken and crenated, but in dilute urine they become larger and their biconcave shape changes to a more spherical one. Normal urine contains no more than 3 red cells/mm³ of uncentrifuged urine, or less than 1 per high power field (hpf) of centrifuged urine. They may be confused with droplets of oil from fingers or catheter lubricant. However, oil droplets are easily differentiated by their variable size and their higher refractive index and they are more circular. Small numbers of red cells do not discolour the urine, and may not give a positive chemical test for haemoglobin (Haemastix), especially if the urine is fresh.

The detection of microscopic haematuria may be very important in diagnosis, particularly in patients with subacute bacterial endocarditis.

Leucocytes (Plate VI)

Leucocytes or pus cells are slightly larger than red cells and can generally be recognized by their round shape, their lobed nuclei and their refractile granular appearance. Their structure is more easily seen if the urine is acidified with a few drops of glacial acetic acid (which will cause the red cells to disintegrate), but without the use of phase contrast microscopy or of special stains they cannot be differentiated reliably from renal tubular epithelial cells. They tend to degenerate rapidly and must be sought in fresh urine. More than 10 leucocytes/mm³ of uncentrifuged mid-stream urine is abnormal in adult women, between 3 and 10 being of doubtful significance; more than 3/mm³ is abnormal in men. This difference between men and women is a result of contamination of the urine by vaginal secretions. Centrifuged urine should not contain more than approximately 5 leucocytes per high power field.

When numerous, pus cells may appear in clumps, and they may form casts. An increase in their numbers suggest that urinary infection may

be present, but infection may occur without such an increase. An increase may also occur without infection being present, particularly if there are renal calculi. Pus may form a deposit visible to the naked eye in the specimen glass, but a certain diagnosis can only be made by seeing pus cells under the microscope.

Epithelial cells

Transitional epithelial cells from bladder or ureters appear as large oval cells with single nuclei. Sheets of polygonal squamous cells come from urethra or vaginal secretion, and if present in quantity in urine collected from women suggests that the specimen is contaminated and probably unsuitable for culture.

Spermatozoa

Spermatozoa occur at times in urine from males and females where their characteristic appearance makes it easy to recognize them. They have no pathological significance.

Prostatic threads

These are found when there is chronic inflammation of the prostate, especially after gonorrhoea. They are much larger than casts, being visible readily enough to the naked eye as they float in the urine or on its surface.

Casts (Plate VII)

The precipitation of a mucoprotein in renal tubules is thought to result in the formation of hyaline casts. On this basic material erythrocytes (forming red cell casts), leucocytes (forming white cell casts), or tubular epithelial cells (forming epithelial casts or, if they contain fats, fatty casts) may be deposited. It is thought that granular casts and waxy casts result from progressive disintegration of the cellular elements of the casts, and that the very broad casts (called 'renal failure' casts) are formed in tubules which are large and dilated through severe parenchymal disorganization.

Hyaline casts themselves are normal. The finding of more complex casts (red cell, white cell and epithelial) indicate that the cells concerned come from the kidney and therefore that the kidneys are diseased.

Casts are easily missed if the urine has been centrifuged too hard and too long, or if the microscopic illumination is too bright. They should be looked for towards the edges of the cover-slip. They are distinguished from other objects which may be mistaken for them—such as hairs, wool, cotton, masses of urates, prostatic threads, rolled-up epithelial cells and so-called 'cylindroids'—by their shape and sharply defined

outline. They are always cylindrical in shape and may have rounded ends, or one end may be ragged as if fractured. Granular casts contain fine or coarse granules. Hyaline casts are pale, transparent and homogeneous, and may be difficult to distinguish from the background, unless phase contrast microscopy is used.

Cylindroids, which resemble casts but are extremely long and narrow and usually tapered, flattened or frayed at the ends, are of no importance.

Micro-organisms

Bacteria may be revealed by Gram-staining the deposit from a centrifuged specimen of urine. They can only be found in this way if present in very large numbers, and if found in a fresh mid-stream specimen of urine this finding strongly suggests a urinary infection. Urine from women may contain *Trichomonas* or yeasts which usually result from vaginal contamination. The former are pear-shaped or round parasites about twice the size of leucocytes; unipolar flagellae may be seen with difficulty. Yeasts are slightly smaller than red cells, and may be confused with them, with air bubbles, or oil droplets.

Bilharzia

Schistosoma haematobium may be found in the urine. The ova (Plate VIII) measure 0.12 mm by 0.44 mm. A spine projects at one pole. The ova of *Schistosoma mansoni*, which are found in the faeces, have a lateral spine.

BACTERIOLOGICAL EXAMINATION

Collection of samples

A mid-stream urine specimen, collected after the vulva or glans penis has been cleaned with tap water, is suitable for most bacteriological purposes. Antiseptic solutions should not be used, for enough antiseptic to interfere with bacteriological culture may get into the urine.

In women the labia are separated by patient or nurse and the vulva is cleaned twice in an anteroposterior direction using swabs soaked in tap water, then finally with a dry swab. Whilst the labia are still held apart some urine, perhaps 20–50 ml, is passed into a toilet or bowl, and the next portion is collected into a clean wide mouthed or plastic jar. Urine collected in this way is unsuitable for culture if collected when the patient is menstruating heavily, but reasonably satisfactory specimens can be collected after insertion of a vaginal tampon if menstruation is light.

Sometimes it is important to obtain a specimen free from urethral contaminants. Provided the bladder is enlarged well above the symphysis pubis (best detected by percussion in adult non-pregnant women) it may be collected by suprapubic aspiration of the bladder. The suprapubic area is shaved and cleaned with antiseptic. An 'intramuscular' needle is inserted at right angles to

the skin surface immediately above the symphysis pubis and urine may be aspirated. It may be necessary to use a longer needle if the patient is fat. The technique is safe and may be used in pregnant women and infants.

The most satisfactory urine specimen for culture is that collected first thing after arising from sleep, for any bacteria in the bladder have been able to multiply undisturbed for several hours. This should be cultured as soon as possible after collection (and certainly within 2 hours) or refrigerated at 4°C immediately after collection, in order to prevent contaminant bacteria from multiplying so much that they may be misinterpreted as pathogens. Mid-stream urines should be cultured quantitatively.

Bacteriological results

In general it is likely that the finding of over 100 000 bacteria/ml of mid-stream urine indicates the presence of urinary infection. However, contamination without infection may sometimes produce counts above this figure: and urinary infection may sometimes be present with lower counts. It is therefore advisable to culture several mid-stream specimens and to discuss the results in doubtful cases with the bacteriologist concerned.

In such cases culture of a suprapubic aspirate may be indicated. Any growth in such a specimen, other than a few skin contaminants, indicates the presence of urinary infection.

ESTIMATION OF RENAL EFFICIENCY

One of the main functions of the kidneys is to rid the body of the waste products of metabolism, which are presented to them in the blood stream. This they do by the excretion of waste products into the urine and by the selective retention of those substances which are not waste. The former is carried out primarily by the glomeruli, which filter off all the contents of the plasma except those of high molecular weight. The tubules then reabsorb certain solutes, excrete others and reabsorb most of the filtered water. Progressive damage to the kidney by chronic renal disease is usually associated with destruction of or cessation of function in a large number of glomeruli. This leads to a reduction in the overall glomerular filtration rate which, if marked, causes a rise in the plasma urea. Under these conditions there is a much greater flow of urine through the remaining tubules, which overloads the diluting and concentrating mechanisms at the distal ends of the tubules. The result is a progressive diminution in the ability of the kidney to concentrate and dilute urine as renal destruction progresses, until finally the specific gravity becomes fixed at 1010.

Plasma levels of urea and creatinine

The plasma level of urea depends on a balance between its production from exogenous and endogenous protein and its excretion by the

kidneys. Its plasma level thus varies with protein intake and may also be raised in conditions such as fever, and haemorrhage into the gastrointestinal tract, which increase endogenous production, but where there is no renal involvement.

In spite, however, of theoretical objections it is widely used by clinicians as a crude indicator of renal function and high levels are found to correlate fairly well with the clinical picture of 'uraemia'. The normal range is usually given as 15–40 mg/100 ml (2.5–6.6 mmol/litre) but levels up to 50 mg/100 ml are not always pathological. Raised levels may be due to 'extrarenal uraemia' in conditions such as dehydration and heart failure. In urinary obstruction and in severe renal failure levels up to 300 or 400 mg/100 ml may be found.

Creatinine (normal 0.7–1.4 mg/100 ml or 62–124 μ mol/litre) is derived almost entirely from endogenous sources, but is more difficult to measure accurately. It is raised in any condition which produces a fall in the glomerular filtration rate and levels as high as 20 mg/100 ml may be reached in advanced renal failure.

For research purposes and sometimes for clinical purposes, it may be necessary to measure impairment of renal function short of that which produces a rise in plasma urea; and to measure individual renal functions. Numerous methods are available and the subject is a complicated one. We shall describe here the estimation of the glomerular filtration rate (GFR) and tests of the renal concentrating and renal acidifying ability.

Estimation of glomerular filtration rate (GFR)

The standard method of estimating the GFR is that of inulin clearance. The renal clearance of a substance is the smallest volume of plasma from which the amount of that substance excreted in the urine each minute could have been obtained at the time of the test. It is calculated from the expression UV/P , where U is the concentration of the substance in the urine (in mg/ml), V is the volume of urine produced (in ml/minute), and P is the plasma concentration of the substance (in mg/ml).

If a substance is excreted by glomerular filtration, and is neither excreted nor reabsorbed during its passage down the tubules, its clearance is equal to the glomerular filtration rate. Although inulin clearance is considered to be an accurate measure of GFR there are practical difficulties in performing the test, and other methods are preferred for routine use.

The clearance of endogenous creatinine is a sufficiently good index of GFR to be popular for this purpose, but it over-estimates GFR at low filtration rates, and in patients with the nephrotic syndrome. In order to minimize inaccuracies due to errors in the timing of urine collections, such collections should be made over 24 hours. It is not necessary to

catheterize patients for this test. Urea clearance is an inaccurate indication of the GFR and should no longer be used for this.

GFR can also be estimated by methods which do not require urine collections. It can be derived mathematically from the plasma creatinine concentration; and the rate of disappearance of $^{51}\text{Co-EDTA}$, or of certain other isotopes, from the circulation may be used to measure it accurately. The normal figure of about 120 ml/minute may be reduced by about two-thirds before the plasma urea concentration becomes elevated.

Renal concentrating ability

The concentration of the urine, but measured as its osmolality, may be as low as 50 mosmol/kg after water loading, or as high as 1300 mosmol/kg after fluid deprivation. Specific gravity, as discussed earlier, is affected by the nature as well as by the number of osmotically active particles in solution. If, however, a random specimen of urine has a specific gravity greater than 1018 it can be assumed that concentrating ability is normal. Fluid deprivation for prolonged periods, or injection of vasopressin, may be used to provide more accurate tests of concentrating ability; a urinary osmolality of at least 750 mosmol/kg, or a specific gravity of 1020 should be attained if concentrating power is normal. In order to produce maximum concentration fluid depletion for 24–36 hours may be necessary. This is unpleasant and may result in excessive dehydration if concentrating power is poor; it is a slightly more effective stimulus to concentration than vasopressin.

Renal acidifying ability

If the pH of a random specimen of urine is below 5.5, it can be assumed that renal acidifying ability is normal. In chronic glomerular disease the urine pH can be reduced below 5.5 until advanced renal failure has occurred. In certain tubular diseases the urine cannot be adequately acidified, even when glomerular filtration rate is normal. In order to test for this, ammonium chloride capsules (0.1 g/kg body weight) are given orally; normally the urine pH is reduced to less than 5.4. The test should not be carried out if the patient is already acidotic. Renal tubular acidosis is a cause of renal calculi, the investigation of which is the most frequent reason for the test being performed.

OTHER INVESTIGATIONS

Excretion urography

This depends on the excretion by the kidney of certain radiopaque organic compounds of iodine. After a plain film of the whole abdomen has been taken, the contrast medium is injected intravenously, and

X-ray films of the kidneys, ureters and bladder are taken after various time intervals, the last after the patient has emptied the bladder. This investigation provides evidence concerning the anatomy and function of the kidneys. It is important in the investigation of suspected urinary obstruction and other surgical abnormalities and is part of the routine investigation of established hypertension. If renal function is poor, then kidneys are more easily seen if tomographic cuts are used.

Micturating cystography

In this investigation similar radiopaque dye is instilled into the bladder, through a catheter passed with strict aseptic precautions. Cineradiograms are taken of the bladder, its outflow tract and the ureters, as the patient voids the dye. This may be important in the investigation of urinary infection or obstruction of the bladder outflow tract.

Cystoscopy and urethroscopy

The interior of the bladder and urethra may be inspected through a cystoscope and urethroscope respectively. The main value of these investigations is in the diagnosis of tumours of the lining epithelium of the bladder and of pathological changes in the prostate. Through the cystoscope it is possible to insert fine catheters into the ureters enabling urine from the kidneys to be collected separately; and by the instillation of contrast medium X-ray films can be taken to show the ureters, pelves and calyces.

Renal biopsy

By means of a special needle inserted into the back, biopsy specimens of the kidney may be obtained. Considerable skill is required and there is a small but definite risk to the patient. The method is of most valuable in predicting the likely outcome and response to steroid therapy of certain patients with nephrotic syndrome. It may also be valuable in the investigation of acute renal failure, and in the diagnosis of systemic conditions such as amyloidosis, sarcoidosis and systemic lupus erythematosus.

5

THE CARDIOVASCULAR SYSTEM

Anatomical Landmarks — Arterial Pulses — Inspection and Palpation — Percussion — Auscultation — Electrocardiography — Radiographic Examination of the Heart — Cardiac Catheterization and Angiocardiography

ANATOMICAL LANDMARKS

The *praecordium* is a term used loosely to indicate the portion of the anterior aspect of the chest which overlies the heart.

It is often necessary to define the exact situation of a point on the front of the thorax, and certain landmarks, some natural and some artificial, are commonly used for this purpose.

The ribs and interspaces on either side form convenient horizontal landmarks. In order to count them, feel for the ridge which marks the junction of the manubrium with the body of the sternum, known as the angle of Louis, or sternal angle. When this has been found, run the finger outwards until it reaches the second costal cartilage, which articulates with the sternum at this level. The space immediately above this is the first intercostal space. The spaces should then be counted downwards well away from the sternum, where they are more easily felt.

In order to define the distance of any given point from the mesial sagittal plane of the body, a series of vertical lines may be drawn on the chest. These are the *midclavicular line*, defined as the vertical line dropped from the centre of the clavicle, or, what amounts to the same thing, the line midway between the middle of the suprasternal notch and the tip of the acromion; and the *anterior, mid-, and posterior axillary lines*, descending respectively from the anterior border, the centre and the posterior border of the axilla.

In the examination of the cardiovascular system one should observe first the arterial pulses, then the blood pressure, then the venous pulse in the neck and finally the praecordium. The usual methods are inspection, palpation, percussion and auscultation. In practice, inspection and palpation are often combined.

ARTERIAL PULSES

The presence of the main peripheral pulses, the radial, brachial, carotid, femoral, popliteal, posterior tibial, and dorsalis pedis pulses should be noted and the volume compared with the other side.

The following observations should then be made:

1. Rate of pulse.
2. Rhythm.
3. Character.
4. Volume.
5. Condition of vessel wall.
6. The presence or absence of delay of the femoral pulses compared with the radials.

To assess the rate and rhythm of the pulse the radial pulse at the wrist is usually used. It is best felt with the tips of the fingers, the patient's forearm being pronated and the wrist slightly flexed (and not in the manner illustrated on the crest of the Royal College of Physicians of London). A central pulse, in either the carotid or the brachial artery, is preferable for studying the character of the pulse or wave-form of the pulse.

Estimates of the 'tension' of the pulse, that is of the blood pressure within the vessel, by palpation, are quite unreliable. The blood pressure should be determined with the sphygmomanometer. The terms 'good', 'bad', 'strong' and 'weak' in relation to the pulse lack precision and should be avoided.

The *rate* of the pulse is stated as so many beats a minute. It is counted, not when the fingers are first laid upon the pulse, but when any quickening due to nervousness of the patient has subsided and the pulse has resumed its normal rate. Count the beats for not less than half a minute. In cases of atrial fibrillation, the pulse rate counted at the wrist may not indicate the true rate of ventricular contractions. In all such cases, the rate of the heart beat should be counted by auscultation at the apex, and the difference between this rate and the pulse rate at the wrist should be recorded. This difference is referred to as the *pulse deficit*. The pulse rate is increased during exercise, in fever and in thyrotoxicosis. It is also increased in paroxysmal atrial tachycardia, atrial fibrillation and atrial flutter (see Electrocardiography, p. 116). It is slowed somewhat in myxoedema, and in complete heart block it beats at a steady 20-40 beats per minute.

Decide next whether the *rhythm* is regular or irregular. If it is irregular, decide if it is completely irregular, whether the irregularity has a recurring pattern, or whether an otherwise regular rhythm is occasionally interrupted by some slight irregularity. The pulse of atrial fibrillation is completely irregular. The irregularity is usually obvious when the

rate is rapid, but becomes less easy to recognize when the rate has been slowed by digitalis. If the rhythm has a recurring pattern, or there are occasional irregularities, these are likely to be due to extrasystoles. An extrasystole is a beat which occurs prematurely, is small and is followed by an unduly long pause. Disorders of rhythm are described more fully under Electrocardiography (p. 118).

Study the *character or form* of the individual pulse wave. It is not usually possible to detect the waves of the normal pulse, or slight variations from the normal, but in certain diseases the character of the pulse is detectably abnormal. The most important of these are as follows:

Anacrotic pulse (the term is a derivative of anadicrotic, meaning two up-beats). This occurs in aortic stenosis, which gives rise to a slow ejection of blood from the left ventricle. The resulting pulse wave has a slow upstroke, an anacrotic wave on the upstroke, and the pulse is of small volume (Fig. 5a).

Collapsing (water-hammer) pulse. This is characterized by a rapid upstroke and descent of the pulse wave. It occurs when there is an abnormal leak from the arterial system, for example aortic regurgitation, patent ductus, arteriovenous fistula, etc. It is best felt when the patient's arm is elevated and the wrist grasped with the palm of the observer's hand against its palmar surface (Fig. 5b).

Bisferiens pulse. This is a combination of the anacrotic and collapsing pulses occurring in combined aortic stenosis and incompetence. The anacrotic wave is of the same height as the percussion wave, and both can be felt distinctly.

Pulsus paradoxus. The pulse becomes smaller, or even disappears, at the end of inspiration. In normal subjects the variation does not exceed 10 mmHg in the systolic pressure. When the decrease is greater it suggests constrictive pericarditis, or pericardial tamponade but also occurs with severe airway obstruction.

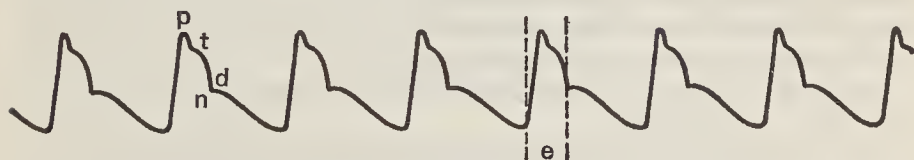


Fig. 4. A normal arterial pulse tracing. p, percussion waves; t, tidal wave; n, dicrotic notch; d, dicrotic wave; e, period of ventricular systole (aortic valve open).

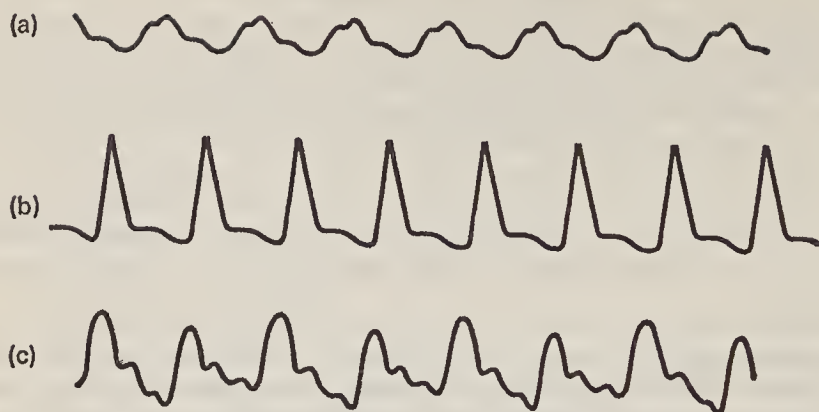


Fig. 5. Arterial pulse tracing showing typical form of the pulse waves in (a) aortic stenosis, (b) aortic incompetence and (c) pulsus alternans.

Pulsus alternans (Fig. 5c). When the ventricle beats strongly, then weakly, in successive beats of normal rhythm, alternation is present. In the radial tracing are seen alternate large and small beats, which are, however, *equidistant*. The condition is often discovered when the systolic blood pressure is taken, and the rate of sounds suddenly doubles as the pressure in the cuff falls. When this condition is discovered, provided the heart rate is moderate and no abnormal rhythm is present, it may be inferred that the heart muscle is severely damaged.

Estimate the *volume* of the pulse beat, that is, the amplitude of movement of the vessel wall during the passage of the pulse wave. Provided that the arterial wall is normal, this is a measure of the pulse pressure. If the vessel becomes rigid, however, the pulse-pressure may widen whilst the pulse volume on palpation may appear normal.

Examine the *condition of the vessel wall*. Sufficient pressure should be exerted on the brachial artery to abolish pulsation in the radial vessel which should then be rolled beneath the fingers against the underlying bone. In young persons, the arteries cannot be felt or are soft. In older persons, they are more easily palpable. In arteriosclerosis they may feel hard like whipcord and may be tortuous.

Delay of the femoral compared with the right radial pulse is found in coarctation of the aorta.

The *typical pulse* of a healthy adult man should be described in the following terms. The rate is 70/minute. The beats are regular in rhythm and equal in volume. The pulse is of normal volume and the arterial wall is just palpable but is neither thickened nor tortuous.

The blood pressure

Certain details are important in the use of the *sphygmomanometer*. The patient should be sitting or lying at ease. The manometer is placed so as to be at the same level as the observer's eye. All clothing should be removed from the arm. The cuff should be applied closely to the upper arm, with the lower border not less than 2.5 cm from the cubital fossa.

The radial pulse is palpated while the cuff is inflated to a pressure of 30 mmHg above the level at which radial pulsation can no longer be felt. The stethoscope is then placed lightly over the brachial artery. The pressure in the cuff is lowered, 5 mmHg at a time, until the first sound is heard, which is the systolic pressure. Continue to lower the pressure in the cuff until the sounds become suddenly faint or inaudible: this is the diastolic pressure.

Precautions. Arterial pressure shows temporary variations with change of posture, after meals, on exertion and notably on excitement. Hence it should be observed only after the patient has been reassured and when he is quietly resting, free from excitement and with the arm relaxed. In nervous patients the first reading is often too high and should be rejected; a second reading will more closely represent the true pressure. The pulse rate at the time should be noted, for blood pressure varies to some extent with the rate of the heart. It is essential to work as quickly as is compatible with accuracy, for compression of a limb itself induces a rise in blood pressure. To reduce this source of error when successive estimations are to be made, the air pressure in the armlet should always be allowed to fall to zero as soon as each reading has been taken. It is important to take the blood pressure of patients taking hypotensive drugs or otherwise suspected of postural hypotension in both the recumbent and the standing positions.

Occasionally the sounds disappear at a point below 200 mm for a period and then reappear, finally disappearing at the point of diastolic pressure. Thus the sounds may first appear when the mercury falls to 210 (systolic pressure), disappear from 180 to 160 (silent gap), reappear and finally disappear at 120 (diastolic pressure). This phenomenon of a *silent gap* is found in certain patients with hypertension; its significance is unknown, but its occurrence makes it important that the armlet pressure should always be well raised at the beginning of an estimation of blood pressure.

It is important to check that the width of the cuff is correct. For an adult, the standard cuff width is 12.5 cm. If a narrower cuff is used, the recorded pressures will be falsely high.

For children, there is a variety of cuffs of different widths. One should

select the size which covers most of the upper arm but leaves a gap of 1 cm below the axilla and above the antecubital fossa.

Occasionally it becomes necessary to compare the systolic blood pressure in the arm with that in the leg. The patient lies face downwards and an 18 cm cuff is applied above the knee and auscultation carried out over the popliteal artery. In coarctation of the aorta, the blood pressure in the legs is lower than in the arms.

Normal blood pressure. The average systolic pressure in healthy adults is 100–140 mmHg, the average diastolic pressure, 60–90 mmHg. In children it approximates to the lower figure in each case, and in the elderly it reaches or even exceeds the higher figure. The difference between the systolic and the diastolic pressures—the pulse pressure—is 30–60 mmHg.

Abnormal blood pressure. A high systolic blood pressure with a normal diastolic pressure (systolic hypertension) is frequently encountered in the elderly, and is a function of inelasticity of the arteries (arteriosclerosis).

A raised diastolic pressure is of much greater significance, and should lead to a search for a primary cause such as renal disease, Cushing's syndrome, phaeochromocytoma, etc. If these causes are excluded, a diagnosis of idiopathic (or essential) hypertension is made.

INSPECTION AND PALPATION

The patient should be examined in a good light, sitting up at an angle of 45° if this is possible. The observer should first note the presence or absence of dyspnoea and cyanosis and the shape of the chest and praecordium. He should then turn his attention to:

1. The neck veins.
2. Veins on the chest wall.
3. The cardiac impulse.
4. Other pulsations.
5. Thrills.

The neck veins

The neck veins communicate directly with the right atrium. Changes in the mean pressure within them, together with the pulsations that occur with each cardiac cycle, give direct information, therefore, about mean pressure in the right atrium and about pressure changes during the cardiac cycle.

The neck veins should be examined with the patient in a good light, and reclining at an angle of about 45°. The neck should be supported so

that the neck muscles are relaxed. The veins normally show slight pulsation, and two or three small waves can be distinguished in each cardiac cycle. There is, however, a mean level, and the perpendicular height of this level above the right atrium indicates the mean hydrostatic pressure within the right atrium. In health this level is the same as that of the manubrium sterni, whatever the position of the subject. The manubrium is therefore a convenient reference point for measuring or estimating the right atrial pressure. This means that in a healthy person reclining at an angle of 45° , the mean level will be invisible, because it is below the clavicle, but some slight pulsation may appear above the clavicle.

Arterial pulsation may also be visible in the neck and has to be dis-

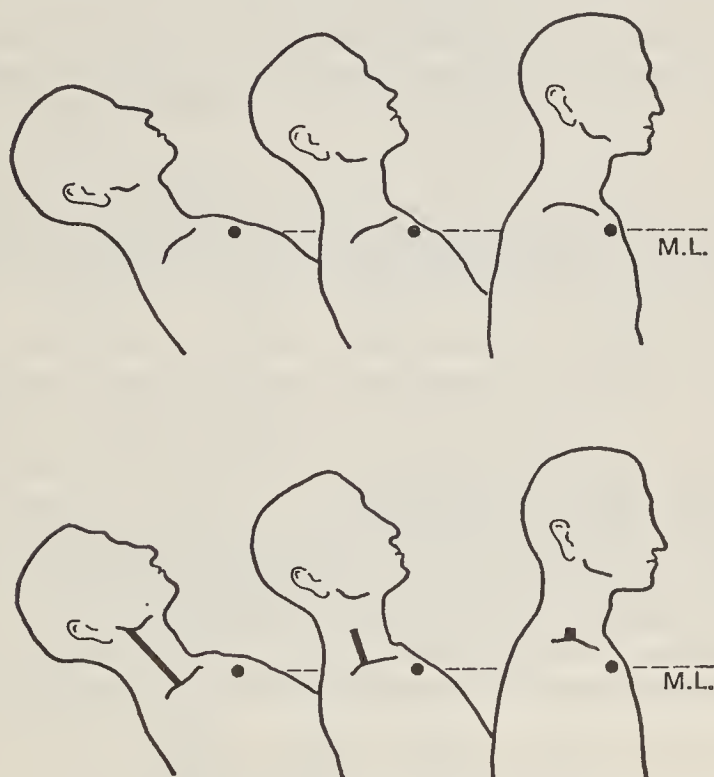


Fig. 6. In health the height of a column of blood in the jugular vein is about evel with the manubrium sterni (ML) whatever the position of the patient. In heart failure, when the right atrial pressure is increased the vertical height of this column is increased, and is above the level of the manubrium sterni whatever the position of the patient.

tinguished from venous pulsation. The venous pulse has a definite upper level, though it may be necessary to sit the patient up higher or lay him lower to find it. This level falls during inspiration when blood is drawn into the heart. Firm but gentle pressure on the abdomen will raise the level by increasing the venous pressure generally. Venous pulsation is usually more sinuous and less sharp than arterial pulsation. Finally it is impalpable, or even when grossly abnormal, e.g. in tricuspid regurgitation, it is only just palpable, while arterial pulsation is easily palpable and thrusting in character.

A raised venous pressure is usually indicative of right heart failure. Occasionally it is due to obstruction of the superior vena cava, in which case the normal pulsations of the venous pulse are absent. A slight rise in venous pressure also occurs with an increase in the circulating blood volume, as in pregnancy and acute nephritis.

No cardiac case has been properly examined till the level of the venous pressure in the neck has been determined. This level is usually seen quite easily by the methods described, but occasionally difficulties occur. The mean venous pressure may be so high, for instance, that the pulsation is obscured behind the jaw when the patient is semirecumbent, and visible only high in the neck when he sits upright. Further, too much reliance should not be placed on the external jugular veins, as these may only be superficial for part of their course. Also kinking of the external jugular vein may cause distension of the vein above the true venous pressure. Internal jugular pulsation is a more reliable guide to the venous pressure.

The early studies of the venous pulse were made with the polygraph, which consists of a tambour recording on paper which is moved by a clockwork mechanism. A simultaneous trace of the arterial pulse was used for timing purposes. The use of this technique at the beginning of this century really marks the advent of modern cardiology.

The venous pulse has three positive waves, *a*, *c*, and *v*, and two negative waves or descents, *x* and *y*. The *a* wave is due to atrial contraction. This is followed by the *x* descent, which is interrupted by a small *c* wave (which is rarely visible on inspection of the neck veins). The *c* wave coincides with the onset of ventricular systole and results from tricuspid valve closure. The *v* wave indicates a passive rise in pressure as venous return continues while the tricuspid valve is closed. When the tricuspid valve opens, blood enters the

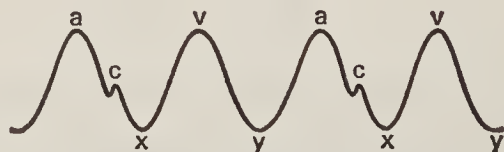


Fig. 7. The venous pulse.

right ventricle rapidly and there is consequently a lowering of the right atrial pressure—the y descent.

The a wave is prominent when the resistance of the right ventricle to filling is increased, as in tricuspid stenosis, or from hypertrophy of the right ventricle due to increased pressure work, in pulmonary stenosis or pulmonary hypertension. In tricuspid regurgitation, the v wave is replaced by a large *systolic* wave due to regurgitation of blood into the right atrium during right ventricular systole. This regurgitant flow of blood may well exceed the forward flow through the heart, and explains why the venous wave in tricuspid incompetence has such a surging expansile character.

Obviously a waves will disappear when there is no active atrial contraction in the atrial fibrillation. In complete heart block (complete atrioventricular dissociation) regular a waves can be seen in the neck while carotid pulsation occurs at a slower independent rate. From time to time atrial contraction occurs when the atrioventricular valves are closed during ventricular systole. All the force of right atrial contraction is transmitted into the veins, to give a 'cannon wave'. If the arterial pulse is abnormally slow, the neck veins should be carefully examined for more rapid, regular a waves and cannon waves. 'Cannon waves' are also seen in nodal rhythm when the atrium and ventricle are activated simultaneously, and the atrium contracts on closed atrioventricular valves.

Veins on the chest wall

The veins of the thoracic wall may be unduly conspicuous. This occurs (a) when the patient's skin is unusually transparent; (b) when an intra-thoracic growth or aneurysm obstructs the return of blood to the heart; or (c) when, in consequence of portal obstruction or obstruction of the inferior vena cava, the blood returning from the abdominal viscera or lower limbs is forced to find its way through collateral channels.

The cardiac impulse

It is customary to locate the apex beat, which is the lowest and outermost point of definite cardiac pulsation, and to locate its position in terms of the particular intercostal space and distance from the midline in which it is felt. The normal position of the apex beat is 9 cm from the midline, or 1 cm internal to the mid-clavicular line in the fifth intercostal space.

The position of the apex beat is a valuable physical sign, if its limitations are understood.

First it must be felt with the patient sitting or lying quite straight. The apex beat of a normal person may be felt in the anterior axillary line, if the person is lying on his left side.

Secondly, it must be realized that the commonest cause of displacement of the apex beat is a deformity of the thoracic cage, usually scoliosis.

A real displacement of the apex beat may be due to disease of the surrounding viscera, which 'push' or 'pull' it from its usual site. Instances of 'pushing' are found in pleural effusion and pneumothorax, and of 'pulling' in pulmonary fibrosis and collapse of the lung. It may also be due to disease of the heart, when displacement of the apex beat may indicate enlargement, particularly of the left ventricle. Of much greater importance in this connection than the exact position of the apex beat is palpation over the left ventricle, the right ventricle, and the pulmonary artery to determine the character of the cardiac impulse, from which information can be gained about the function of the ventricles.

The *left ventricle* normally produces the apex beat. When it is hypertrophied, the beat becomes more forceful and may extend outwards towards the axilla.

The *right ventricle*, when hypertrophied, can be felt by placing the hand firmly over the praecordium just lateral to the sternum, when a definite 'lift' will be detected.

The *pulmonary artery* can be palpated (when enlarged) in the second left intercostal space on expiration.

If an hypertrophied ventricle is present, an attempt should be made to differentiate between that resulting from obstruction to outflow (systolic overload) and that associated with excess filling of the ventricle (diastolic overload). Systolic overload, due to aortic stenosis or systemic hypertension causes a forceful sustained heave. On the other hand, the excessive diastolic expansion, occurring in aortic and mitral regurgitation, causes diastolic overload, and is followed by easy and rapid ejection of blood from the ventricles, producing an equally forceful but less sustained impulse.

Failure to detect the apex beat in the usual area is usually due to the fact that it is obscured by a rib. One should also remember that it is felt on the right side in congenital dextrocardia.

Other pulsations

In addition to the pulsations already described, movements should be looked for at the root of the neck, the front of the chest, and the epigastrium.

In the suprasternal notch the pulsation is usually systolic in time, and when well marked may be an indication of an unfolded aortic arch in hypertension or coarctation of the aorta or of aneurysm of the arch of the aorta.

In the neck various pulsations may be observed. These may be either arterial or venous. The latter has already been described. The carotids pulsate visibly on exertion, from mental excitement, in diseases which cause overaction of the heart, such as thyrotoxicosis and in cases of

aortic regurgitation, hypertension and aneurysm of the aorta. In hypertension, especially in women, and in association with a high aortic arch, the right carotid sometimes shows abnormal pulsation due to kinking, which must not be mistaken for aneurysm.

In the thorax, a rare source of pulsation in unusual parts is *aneurysm of the aorta*. Such aneurysmal pulsations always manifest themselves at first above the level of the fourth rib, though at a later period they may affect a considerable portion of the chest wall. The position of the impulse varies according to the part of the aorta which is diseased. If the *ascending aorta* is affected, the pulsation is chiefly to the right of the sternum, whilst the *transverse aorta* gives rise to less distinct pulsation under the manubrium sterni, and the *descending aorta* still more to the left. The time of this pulsation is systolic, following immediately on the apex beat, and it may be observed to be expansile in character.

In coarctation of the aorta a collateral arterial circulation develops, and pulsation may be detected in superficial arteries in the chest wall. This may best be seen over the back with the patient bending forward to touch his toes (Plate IV).

Pulsation *in the epigastrium* is most commonly due to nervousness or excitement in a thin person. Less commonly it is due to a hyperkinetic right ventricle, or to transmission of the aortic pulsation by a tumour, such as a carcinoma of the stomach. Occasionally it is due to distensile pulsation of the liver in heart failure with tricuspid regurgitation; and very rarely to an aneurysm of the abdominal aorta, which may be palpable as an expansile swelling.

Thrills

Any sound or murmur which is loud will be palpable. A palpable murmur is called a thrill and transmits to the hand a sensation like the purring of a cat. The character, timing, and variation with respiration of thrills are the same as those of the corresponding murmurs and are discussed with murmurs. An abnormally loud sound is felt as a shock. A third sound or atrial sound is often easier to feel than to hear. The loud first sound of mitral stenosis is often palpable and the sounds of aortic and pulmonary valve closure are often palpable in systemic and pulmonary hypertension respectively.

PERCUSSION

Where radiographs of the chest are readily available percussion of the cardiac dullness is hardly worth pursuing. Where radiographs are not so available percussion may assist in the diagnosis of large pericardial effusions and of aneurysms of the ascending aorta. In the former the whole area of dullness is increased; and in the latter an area of dullness

to the right of the sternum at the level of the second interspace may be detected. In pulmonary emphysema the area of cardiac dullness may be diminished.

AUSCULTATION

To become skilled in auscultation of the heart requires a great deal of practice. Eventually one becomes attuned to the various sounds and murmurs, and learns to focus attention on one portion of the cardiac cycle at a time. It is wise to palpate the carotid artery (rather than the radial) while auscultating, to avoid the common error of mistaking systole for diastole and vice versa.

The stethoscope. Avoid fancy stethoscopes and long tubing. It is helpful to have one which combines both a bell-type chest piece and a diaphragm. High pitched sounds such as aortic diastolic murmurs are heard better with the diaphragm.

Auscultatory areas

It is customary to listen first in the following areas:

The mitral area, which corresponds to the apex beat.

The tricuspid area, which lies just to the left of the lower end of the sternum.

The aortic area, which is to the right of the sternum in the second intercostal space.

The pulmonary area, which is to the left of the sternum in the second intercostal space.

It must be appreciated (*a*) that auscultation must not be confined to these areas, and (*b*) that noises heard in a particular area do not necessarily come from that particular valve; for example murmurs originating at the aortic valve are frequently best heard at the mitral area. There is usually, however, good correlation.

The events of the cardiac cycle are illustrated in Fig. 8. At the onset of ventricular systole, the mitral and tricuspid valves close almost simultaneously to give the *first heart sound*. The opening of the aorta and pulmonary valves occurs next and is inaudible. The closure of the aortic and pulmonary valves gives rise to the two components of the *second sound*. It will be seen that because of the lower pressure in the right ventricle compared with the left, closure of the pulmonary valve follows that of the aortic valve. After a brief period the mitral and tricuspid valves open inaudibly in the normal heart.

Deviations from the normal in disease

In disease the following deviations from the normal may occur:

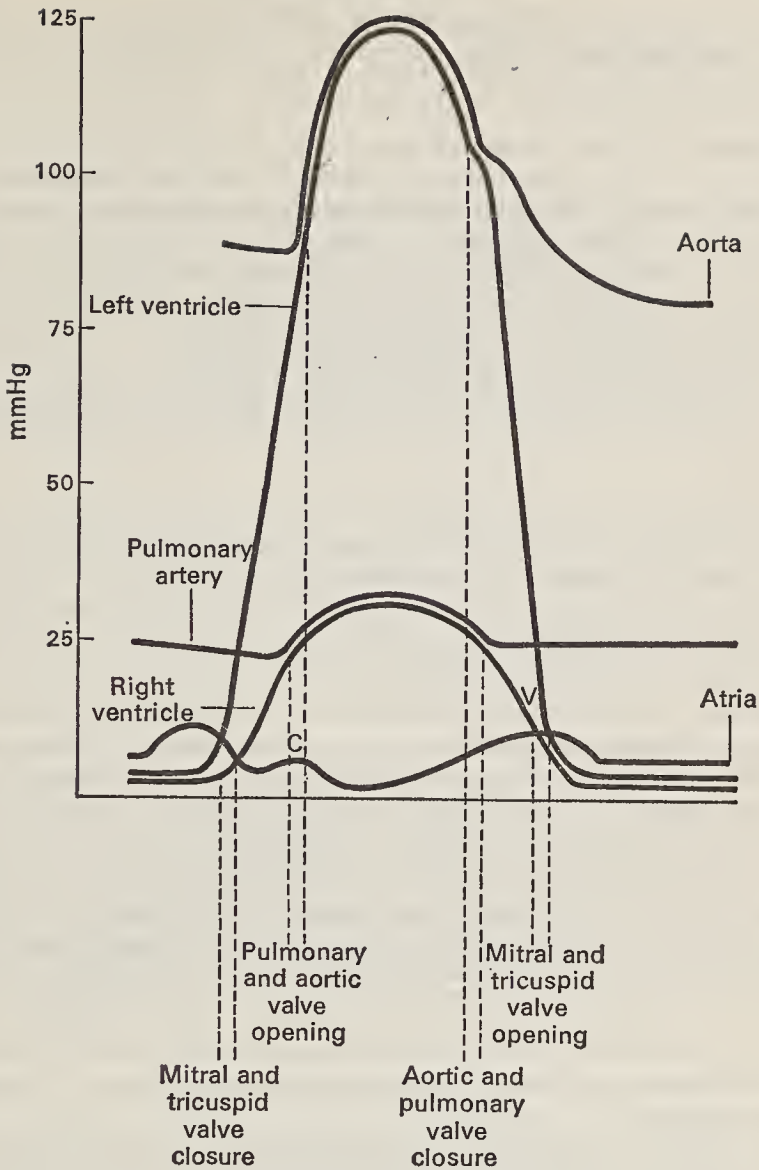


Fig. 8. The cardiac cycle.

1. The sounds may have a different intensity, both absolutely and relatively to each other, from that possessed in health.
2. The sounds may be abnormally split.
3. A triple rhythm may be present.

4. Adventitious sounds may be heard, either replacing or occurring along with the heart sounds.

Alterations in intensity

In patients with thick chest walls, and in those with a serious degree of emphysema, the heart sounds may scarcely be audible, though there is no heart disease. Conversely, in the presence of serious heart disease the sounds may appear quite normal. Thus alterations in the intensity of the heart sounds are significant only when considered in relation to all the other features of the case. The heart sounds are distant or inaudible in pericardial effusion. Accentuation of the first sound is often present in mitral stenosis and in tachycardia from any cause.

An absolute accentuation of the sound of aortic or pulmonary valve closure is found when there is systemic or pulmonary hypertension.

Splitting

The mitral valve closes slightly before the tricuspid valve, and this can give rise to splitting of the first sound. This splitting is difficult to detect by auscultation and even phonocardiography, because both components are very low-pitched and merge one into the other. When it is heard, splitting of the first sound is not a sign of heart disease and is of importance only because its two components may be confused with an atrial sound and a first sound, or with a first sound and a systolic ejection click.

Splitting of the second sound is much easier to hear, because the aortic and pulmonary valve closure sounds (A_2 and P_2) have high-pitched harmonics, and the two sounds can be separated, especially when the diaphragm is used for auscultation. Aortic valve closure (A_2) is audible in all areas. Pulmonary valve closure (P_2) is audible only in the pulmonary area and for a short distance down the left sternal edge, unless its intensity is greatly increased due to pulmonary hypertension. It follows that splitting of the second sound is usually heard only at and close to the pulmonary area. Splitting is most easily heard in children, and may not be audible in older adults, especially men, when muscle noise, a thick chest wall, and emphysema make P_2 inaudible. Normally P_2 always follows A_2 , and the splitting is widest during inspiration and narrowest in expiration, when the two components usually merge to give a single sound. Splitting of 0.06 second during inspiration and 0.02 second (a very close split or single sound) in expiration would be average for a child or young adult.

The mechanism of splitting of the second sound is as follows. During inspiration blood is drawn into the thorax, there is a relative rise in right atrial pressure, and the right ventricular stroke volume increases. The duration of right ventricular systole measured from the first sound to P_2 is increased, and P_2 is therefore slightly delayed. Conversely the left ventricular stroke volume falls during inspiration, because the greater negative pressure within the thorax enlarges the capacity of the left atrium and pulmonary veins and reduces left atrial pressure and hence left ventricular filling and stroke volume. Thus left ventricular systole is shortened and A_2 is earlier. During

inspiration, then, A_2 occurs earlier and P_2 later, so that splitting of the second sound widens. During expiration the changes are exactly opposite and the splitting narrows. Movement of P_2 is considerably greater than that of A_2 . This concept of respiratory variation in right and left ventricular stroke volume is of the greatest importance.

Triple rhythm

Phonocardiography shows that in addition to the two heart sounds generally recognized, a third sound and an atrial sound are often present. When either of these additional sounds are prominent and audible, they give a cadence of sounds known as triple rhythm.

The third sound follows the aortic component of the second sound by about 0.15 second. It is usually best heard in the mitral area and is lower pitched than the second sound, which it follows. Triple rhythm from a third sound is common in healthy young persons. It is an important sign in heart failure from any cause, and may be heard shortly after cardiac infarction. The third sound is attributed to rapid ventricular filling and is found in the relatively hyperkinetic circulation of young persons and where the mitral diastolic flow is increased as in mitral regurgitation and ventricular septal defect. In heart failure the atrial pressure is increased and the early filling of the ventricle is rapid. A third sound is also heard in disease when the distensibility of the ventricular muscle is altered. The sound arises from vibrations in the atrioventricular valve structures and in the ventricular muscle. Theoretically, a third sound could arise on either side of the heart, but in fact it nearly always arises in the left heart.

The atrial sound is a low-pitched sound occurring before the first sound. It is not heard in health. Triple rhythm from an atrial sound can originate in the right heart from pulmonary stenosis or pulmonary hypertension, and in the left heart from systemic hypertension. It is not always a sign of failure but does indicate that the heart is under strain. Like the third sound, it used to be thought to be a 'filling sound', but now a valvular origin is suspected.

When the heart rate is rapid (100 or more), diastole tends to be relatively shortened compared with systole, and a third sound may come to overlie an atrial sound, giving rise to a 'summation gallop'. Usually, when the heart slows, a loud third or atrial sound is audible.

Murmurs

Murmurs have a blowing or musical quality. They are due to turbulence in the blood flow at or near a valve or an abnormal communication within the heart. Not all murmurs are due to organic damage in the heart; they may be due to such causes as an abnormally rapid flow of blood through a normal valve. Such murmurs are called flow murmurs. Murmurs also occur at the site of arterial stenoses. In examining a murmur the following points must be noted:

Its time of occurrence. Murmurs may be (a) systolic, (b) diastolic or (c) continuous throughout systole and diastole. *Systolic murmurs* are

either *pansystolic*, as in mitral and tricuspid regurgitation and ventricular septal defect, or *ejection*, when they arise either at the pulmonary or aortic valves. *Diastolic murmurs* are either *immediate* (or early), starting at the second heart sound and occurring as a result of aortic or pulmonary regurgitation; or *delayed* (or mid), when there is a short gap after the second heart sound and the beginning of the murmur. These murmurs arise at the mitral or tricuspid valves.

The behaviour of the murmur during respiration. The stroke output of the right heart increases during inspiration, while that of the left heart is reduced. It follows that a murmur originating on the right side of the heart will become louder during inspiration. Because the chest wall tends to be carried away from the heart during inspiration, and the lung tends to cover the heart, most cardiac events are not so well heard during inspiration. Any murmur, therefore, which increases on inspiration can be attributed to the right heart and any murmur which increases on expiration can be attributed to the left heart. When the cardiac lesion is severe, however, the variation in stroke volume may not occur with respiration and the murmur is of constant intensity.

Its point of maximum intensity and direction of selective propagation. The maximum loudness of a murmur which has been produced at a given valve usually occurs at the point where the valve sound would be best heard in health. To this rule, however, there are some exceptions.

Murmurs are not equally well heard at all points on the chest wall which are equidistant from the point of their greatest intensity, but each is much more distinctly audible in some directions than in others, i.e. such murmurs have a direction of selective propagation.

The character of a murmur. This also helps to determine its origin. Obstructive murmurs, from obstruction to the onward flow of blood through a narrowed valve, are usually rough; regurgitant murmurs, from leakage backwards through a closed but incompetent valve, are softer and blowing. The loudness of a murmur has no relation to its importance. A very loud murmur may be less significant than one so soft as to be nearly inaudible.

It has already been stated that murmurs are due to turbulence in the flow of blood. The most important cause of turbulence within the heart is the rapid flow of blood through a relatively small orifice, and this means that there must be a considerable difference in pressure on the two sides of the orifice leading to a sufficient gradient. In the normal adult heart, the pressure gradients across open valves are very small, because the valve orifices are fairly large in relation to the flow of blood through them. There are therefore no murmurs. If, however, a valve

orifice should be narrowed by disease, a sufficient pressure gradient may build up at certain times during the cardiac cycle to produce a murmur.

In the next paragraphs the haemodynamics in some common forms of valvular disease are presented, in order to show how murmurs are produced and why they arise when they do. Certain abnormal valve sounds which are associated with the opening and closing of abnormal valves will also be mentioned, and a note of the associated clinical findings will be included.

Mitral valve disease

Mitral stenosis

Narrowing of the mitral valve orifice, mitral stenosis, is due to the fusion of the two valve cusps along their margins, extending from the valve ring at the periphery in towards the centre. The normal valve closed by the apposition of 2 mobile cusps over a length of 3.5 cm becomes a fibrotic diaphragm with a small central orifice closed by the apposition of 1 cm or less of cusp tissue.

The haemodynamics in a mild and a severe case of mitral stenosis are shown diagrammatically in Figs 9 and 10.

Loud first heart sound. In normal subjects at the end of diastole the mitral valve is almost closed as ventricular filling is complete. In contrast, with mitral stenosis, the valve remains open at the end of diastole and is forcibly shut by the rise of pressure due to left ventricular systole. The mitral valve therefore closes very rapidly and a loud first heart sound results.

The opening snap of the mitral valve is a loud sound heard just after the second heart sound. It is a consequence of the high left atrial pressure, which forces the mitral valve to open rapidly when the ventricle relaxes. If the mitral valve is rigid and calcified, it follows that the first heart sound will not be accentuated and the opening snap will not be present.

The mitral diastolic murmur is a low-pitched rumbling murmur heard at the apex; it is due to blood passing through the narrowed mitral valve when there is a pressure gradient between the left atrium and left ventricle. (In the normal heart no such gradient exists and the pressures equalize rapidly when the mitral valve opens.)

The presystolic murmur results from atrial systole, and therefore immediately precedes the first heart sound. It is due to an increase in flow across the narrowed mitral valve during atrial contraction. It has been traditional teaching that when atrial fibrillation is present and there is no effective contraction of the atria, there cannot be a presystolic

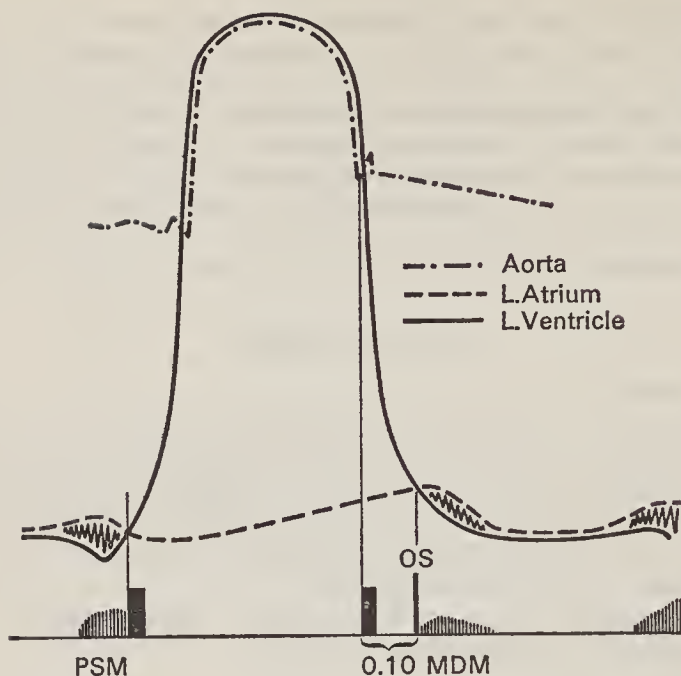


Fig. 9. The relationship of murmurs to haemodynamics in mild mitral stenosis.

murmur; but a slight presystolic accentuation of the diastolic murmur may in fact be heard in the absence of atrial contraction, as the flow continues towards the end of diastole through a partially closed valve.

Mild mitral stenosis, is associated with a slightly raised left atrial pressure. Mitral valve opening occurs at a normal time, so that the opening snap is relatively late and the diastolic murmur is short.

Severe mitral stenosis causes a considerably raised left atrial pressure. The opening snap occurs early and the diastolic murmur is long.

The auscultatory signs in mitral valve disease may be sharply localized at or near the apex. It is always worth while listening with the patient lying on his left side, as this throws the heart up against the chest wall and apical murmurs are better heard. In cases of mild mitral stenosis the patient should be examined after exercise, for this increases the cardiac output and hence accentuates the pressure gradients and the murmurs.

Mitral regurgitation

In mitral regurgitation the cusps of the mitral valve fail to close completely during ventricular systole, which results in a jet of blood being

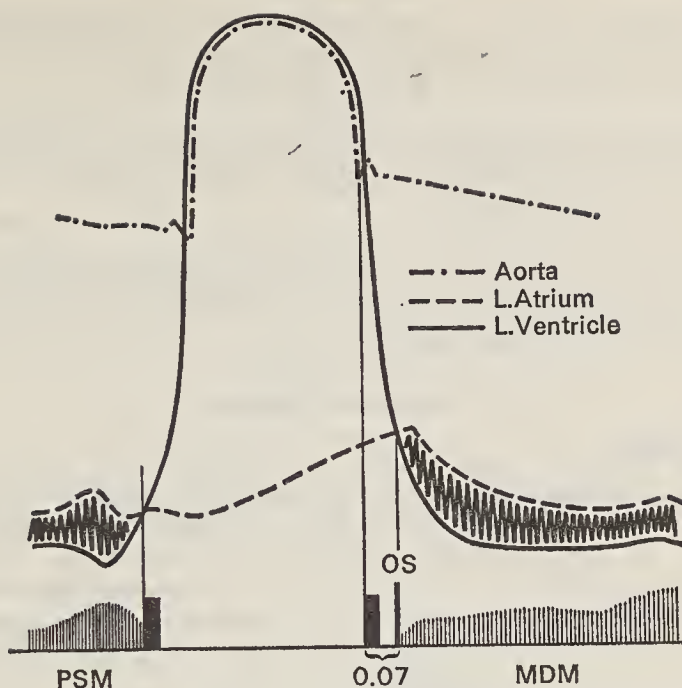


Fig. 10. The relation of murmurs to haemodynamics in severe mitral stenosis.

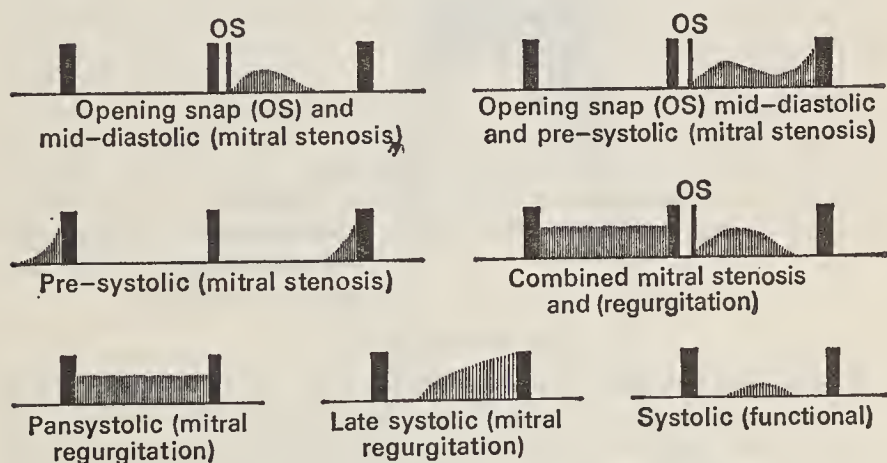


Fig. 11. Mitral murmurs.

forced back into the left atrium throughout the systole, leading to a pansystolic murmur.

The *mitral pansystolic murmur* is due to blood leaking back through the mitral valve in ventricular systole. It starts therefore when the mitral

valve normally closes, i.e. with the first heart sound, and continues throughout systole. It is best heard at the mitral area, and radiates into the axilla and increases in intensity with expiration.

The mitral late systolic murmur. This indicates mitral regurgitation and is usually associated with disease of the chordae tendinae or papillary muscles of the mitral valve.

The soft first heart sound. The bulk of the first heart sound is due to mitral valve closure, and if this closure is imperfect, the resulting heart sound will be softer than normal.

Aortic valve disease

Aortic stenosis

The haemodynamics in aortic stenosis are shown diagrammatically in Fig. 12. There is a considerable pressure gradient between the left ventricle and aorta, and this gradient is greatest in the middle of systole, and is relatively small early and late in systole. The murmur of aortic stenosis is therefore midsystolic. All murmurs due to the ejection

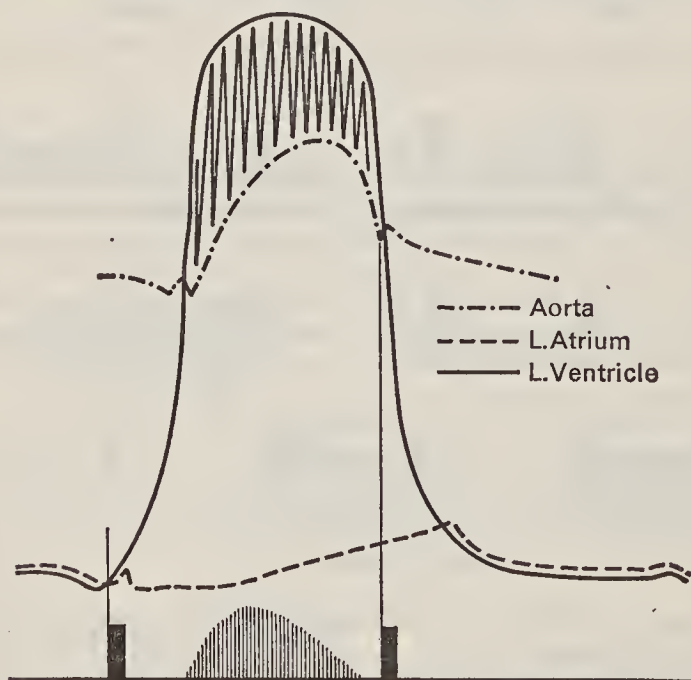


Fig. 12. The relation of murmurs to haemodynamics in aortic stenosis.

of blood through abnormal semilunar valves, or to abnormally high blood flow through normal semilunar valves, have this same pattern and are called 'ejection' systolic murmurs.

The aortic ejection murmur. This murmur is heard in the aortic area and radiates into the neck although it is frequently well heard at the mitral area. It increases on expiration.

The aortic ejection click is due to opening of the aortic valve in aortic stenosis. It is therefore heard just after the first heart sound at the beginning of the ejection murmur.

Delayed and soft closure of the aortic valve are much less easily detectable signs of aortic stenosis. The aortic valve closure is delayed because it takes longer for the left ventricle to expel its blood through the narrowed valve. Pulmonary valve closure may then precede the aortic closure. This is known as 'reversed splitting' of the second sounds. Aortic closure will also be soft simply because the mobility of the semilunar cusps is reduced and they do not snap together with the usual force.

Aortic regurgitation

The aortic leak is surprisingly large, and in severe regurgitation may equal the forward flow of blood into the circulation. That is to say that double the normal stroke output may be ejected through the aortic valve into the aorta during systole, and this may give rise to an aortic ejection murmur. Thus an aortic ejection murmur in a patient with aortic regurgitation does not necessarily indicate a concomitant stenosis.

The early or immediate diastolic murmur starts with the second heart sound and continues for a variable time in diastole. It is a high-pitched murmur, usually soft, and often requires intent auscultation for its recognition. It is best heard down the left sternal edge when the patient sits forward and breathes out, and the examiner listens with the diaphragm of his stethoscope.

Tricuspid valve disease

Organic tricuspid valve disease is almost always of rheumatic aetiology and is almost always accompanied by rheumatic mitral and aortic valve disease. The diagnosis can often be made from inspection of the neck veins (p. 99). The murmurs of tricuspid stenosis and tricuspid regurgitation have the same timing and character as the corresponding mitral lesions, but are best heard at or near the lower end of the sternum and may be louder in inspiration. Tricuspid opening snaps are rare.

Functional tricuspid regurgitation is often found with congestive failure from any cause. It is due to dilatation of the tricuspid valve ring,

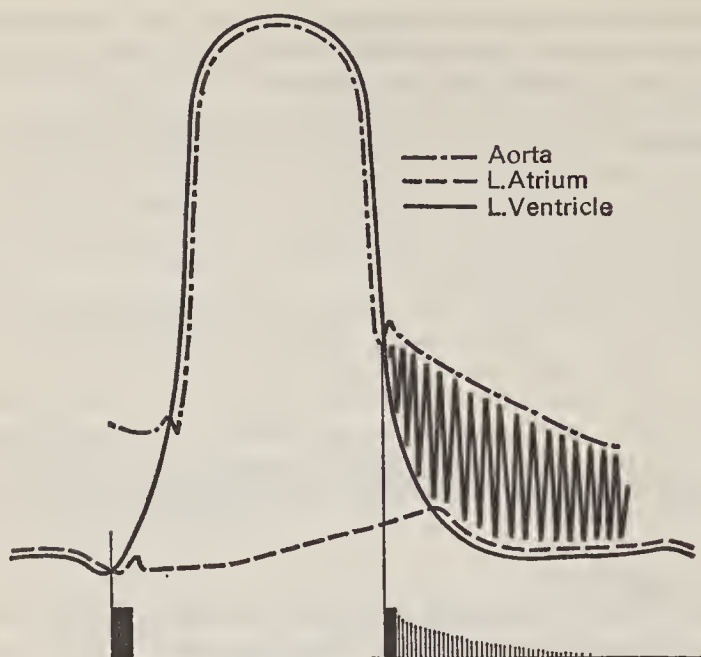


Fig. 13. The relation of murmurs to haemodynamics in aortic regurgitation.

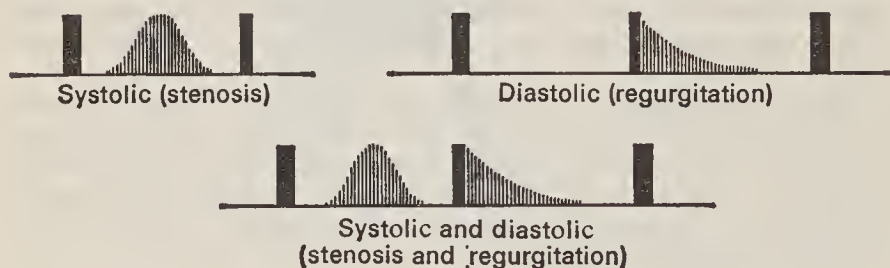


Fig. 14. Aortic murmurs.

consequent on dilatation of the right ventricle, and the characteristic changes in the venous pulse disappear or diminish when the heart failure is controlled.

Congenital heart disease

Pulmonary stenosis

In order to maintain the cardiac output in the presence of pulmonary stenosis the right ventricular pressure has to rise considerably and exceeds the level of systemic pressure in severe cases. In mild pulmonary

stenosis the only abnormal signs are found on auscultation. The ejection systolic murmur (and thrill if present) is most prominent in inspiration and best heard in the pulmonary area. It is usually preceded by an ejection click. The second sound behaves normally on inspiration, but the width of splitting may be wider than normal because of a delay in pulmonary valve closure. If the stenosis is severe, the closure of the pulmonary valve becomes soft or inaudible.

Atrial septal defect

Blood flows through the defect from left atrium to right, since the normal pressure relationships are maintained. The blood flow through the defect is often more than twice the flow entering the left ventricle (i.e. the systemic blood flow). The flow of blood entering the right atrium will then be the systemic flow plus the flow from the left atrium through the atrial septal defect, i.e. three times the systemic flow. This torrential flow passes from the right atrium to the right ventricle, pulmonary artery, lung vessels and so back to the left atrium.

Because the defect is very large and the pressure gradient very small (about 1 mmHg), there is little turbulence and no murmur from blood flowing through the defect. The characteristic auscultatory sign is the relatively wide splitting of the second heart sound, uninfluenced by respiration as the large right ventricular stroke volume does not vary with respiration. The torrential blood flow through a normal pulmonary valve produces a functional ejection pulmonary systolic murmur which is moderately loud. A functional tricuspid flow murmur is often present. It is mid-diastolic, soft and best heard at the lower end of the sternum during deep inspiration.

Ventricular septal defect

Ventricular septal defects are rarely as large as atrial septal defects and usually less than 1 cm in diameter. The right ventricular systolic pressure is normally about one-fifth of that in the left ventricle, so that there is a pressure gradient from left ventricle to right throughout systole.

In mild cases (*maladie de Roger*) where the defect has the cross sectional area of a pencil lead, the only abnormal sign is a pansystolic murmur, loudest in expiration, usually accompanied by a thrill, and best heard at the lower end of the sternum.

In more severe cases there is evidence of increased blood flow through the left heart and blood vessels of the lung. In addition to the pansystolic murmur and thrill there may be a soft short mid-diastolic murmur due to the rapid flow of blood through a normal mitral valve. Splitting of the second sound is normal, though A_2 may be difficult to hear, as it tends to be obscured in the loud pansystolic murmur in small

and moderate sized defects. Where there is pulmonary hypertension the second sounds become fused.

Patent ductus arteriosus

The communication has the same size range as in ventricular septal defect, but its length also tends to limit flow. The pressure gradient from aorta to pulmonary artery is present throughout the cardiac cycle and is greatest towards the end of systole. The increased flow affects the left heart and pulmonary circulation exclusively.

Again, if the communication is small, a murmur is the only abnormal sign. It is best heard at the pulmonary area in expiration, and is called continuous. The accentuation of the continuous murmur about the time of the second sound gives it a particular character, and it is sometimes described as a machinery murmur. In addition to the continuous murmur, a short soft functional mitral diastolic murmur may be present.

A continuous murmur is not specific for patent ductus arteriosus, but can be produced by any arteriovenous communication of the rapid flow of blood through any narrowed vessel. Patent ductus arteriosus is the commonest of several such possible conditions within the thorax. Particular care should always be taken not to confuse it with a venous hum.

Fallot's tetralogy

The tetralogy consists of pulmonary stenosis, ventricular septal defect, right ventricular enlargement and overriding of the aorta, i.e. the aorta arises astride the ventricular septal defect. The essential features are pulmonary stenosis of at least moderate severity and a large ventricular septal defect, so that the pressure in the two ventricles is equalized. The right ventricular output is ejected partly into the pulmonary artery through the pulmonary stenosis, and partly into the aorta through the ventricular septal defect. The admixture of deoxygenated blood with the left ventricular output makes the patient cyanosed to some degree at least. Because the right ventricular pressure cannot exceed the left, as it does in severe pulmonary stenosis with intact ventricular septum, the degree of right ventricular hypertrophy is less.

The auscultatory signs are a pulmonary ejection murmur and a single second sound. P_2 is inaudible because of the low blood flow and low pressure in the pulmonary artery.

Coarctation of the aorta

In this condition there is a stricture of the aorta at or near the insertion of the ligamentum arteriosum. In order to maintain a satisfactory distal circulation, the arterial pressure proximal to the coarctation rises considerably and this leads to elongation of the ascending aorta. Prominent

arterial pulsation behind the manubrium sterni in a young person strongly suggests this diagnosis, and the radial and femoral pulses should be felt simultaneously. Normally the timing of the pulse wave is identical. In coarctation the femoral pulse is both delayed and diminished, or it is absent. The arterial pressure is considerably raised; the left ventricle is readily palpable and hypertrophied; there is an ejection click from dilatation of the aorta, and an aortic ejection systolic murmur, often associated with a congenital bicuspid aortic valve. Collateral vessels linking the subclavian arteries which arise above the stricture with intercostal arteries arising below the stricture can usually be felt above the scapulae, and are well seen with the patient bending forwards in a good light (Plate IV).

Pulmonary hypertension

A rise in pulmonary artery pressure to systemic or near-systemic levels is always associated with narrowing of the pulmonary arterioles either from vasoconstriction or thrombosis and embolism. Pulmonary hypertension may be found in some cases of cor pulmonale, in some cases of septal defect, occasionally with mitral stenosis, or without apparent cause—primary pulmonary hypertension. Whatever the cause, the cardiovascular signs are dominated by the pulmonary hypertension.

There is usually a prominent *a* wave in the neck, the pulse is often very small from the severe obstructive lesion in the pulmonary arterioles, and pulsation over the right ventricle may be palpable. On auscultation there is a loud ejection click, close splitting of the second sound with a very loud and palpable pulmonary valve closure and often a pulmonary diastolic murmur.

Flow murmurs

Systolic flow murmurs are due to an abnormally high flow of blood through a normal valve, which causes turbulence of the blood stream. They are common and in themselves of no serious significance; yet many people have been labelled as having heart disease, simply because they had a systolic flow murmur. By contrast *diastolic murmurs are always significant*.

Systolic flow murmurs are common in infants and young children, and in the elderly. They also occur in any condition such as anaemia, thyrotoxicosis or hypertension, which causes an increased blood flow.

In the adult most flow murmurs are aortic. They are loudest in expiration, heard best in the mitral or aortic area, soft, and never accompanied by a thrill. The other findings in the cardiovascular system

are normal, except where the cardiac output is raised. The distinction between trivial aortic stenosis and an aortic flow murmur is difficult.

Exocardial sounds and murmurs

Venous hum

Sometimes in children a continuous murmur can be heard in the neck and upper chest which is due to kinking and partial obstruction of one of the larger veins in the neck, and interference with continuous flow of blood through the vein. The origin of the murmur should be suspected because of the youth of the patient, and the loudness of the murmur in the neck. The hum can be obliterated by pressure on the neck, which produces complete obstruction of the vein, or by altering the position of the neck so as to relieve the venous obstruction. It is particularly important to exclude a venous hum if a diagnosis of patent ductus is being considered.

Cardiorespiratory murmurs

These murmurs are systolic and due to the rhythmic compression of a lobule of lung by the beating heart. Characteristically these murmurs are loudest at a particular point in the respiratory cycle, disappearing as the patient breathes in a little more or out a little more.

Pericardial friction rubs

These have a superficial 'leathery' quality and have a to-and-fro character, being present both in systole and diastole. This may be sharply localized and vary in position from day to day. Effusion into the pericardium, by separating the pericardial surfaces, may eliminate the rub. Pleural rubs from pleurisy of a portion of lung near the heart may have a similar quality but are much reduced in intensity by having the patient hold his breath.

ELECTROCARDIOGRAPHY

The action of the excitable tissues of the body is associated with electrical activity. Changes in electrical potential associated with the contraction of the heart can be recorded from the body surface.

There must be two points of contact with the body to lead the electrical activity of the heart to the galvanometer. These connections are termed electrocardiographic leads. The leads in common use are:

The standard limb leads (bipolar limb leads)

- Lead I right arm–left arm
- Lead II right arm–left leg
- Lead III left arm–left leg

The unipolar or V leads. The two connections in these leads are (a) an exploring electrode and (b) an indifferent electrode which is produced by joining the limb leads together and thence through a resistance—for all practical purposes it is neutral.

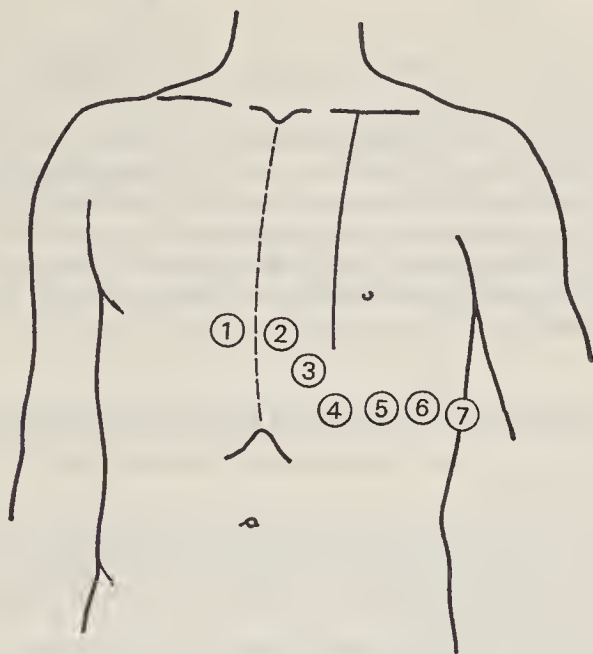


Fig. 15. The position of the exploring electrode for chest leads.

The augmented unipolar limb leads. These are termed:

- aVR The exploring electrode placed on the right arm
- aVL The exploring electrode placed on the left arm
- aVF The exploring electrode placed on the left leg

The unipolar chest leads. The position of the exploring electrode is as follows:

- V₁ 4th intercostal space just to the right of the sternum
- V₂ 4th intercostal space just to the left of the sternum
- V₃ midway between V₂ and V₄
- V₄ 5th left intercostal space in the midclavicular line
- V₅, V₆ and V₇ on the same horizontal line as V₄ in the anterior, mid and posterior axillary lines respectively

The deflections or waves of the electrocardiogram are designated by the letters PQRST as shown (Fig. 16). The P wave is associated with



Fig. 16. The terminology of the electrocardiogram.

atrial excitation, the QRS with ventricular excitation (depolarization), and the T wave with ventricular recovery (repolarization). The Q wave is an initial downward deflection in the QRS complex (Fig. 16).

The PR interval (measured from the beginning of the P wave to the beginning of QRS complex) is normally less than 0.2 second. The duration of the QRS complex is normally less than 0.1 second. The appearance of a normal electrocardiogram is shown in Fig. 27.

Reading and interpretation of the electrocardiogram

An electrocardiogram must be examined systematically. A convenient method is as follows:

1. Determine the cardiac rate and rhythm (see below).
2. Assess the PR interval and the width of the QRS complex.
3. Examine the P wave (atrial contraction) and the QRS complex (ventricular depolarization).
4. Examine the ST segment and T wave (ventricular repolarization).

It is not possible in this text to do more than give some examples of the kind of abnormalities than can be demonstrated in electrocardiograms. Those who wish to understand this subject must refer to specialized textbooks. Considerable experience is required even to be familiar with the limits of the normal tracing. One should also notice that, since many electrocardiographic abnormalities can arise from a variety of causes, they cannot be finally interpreted unless they are considered in relation to the clinical findings.

The electrocardiogram in disorders of cardiac rhythm

By providing together a record of atrial excitation (P waves) and ventricular excitation (QRS complexes) the electrocardiogram has advanced our understanding of the cardiac dysrhythmias. P waves are usually best seen in Lead II or in the right-sided chest leads (V_1) and these leads are therefore most valuable in the disorders of cardiac rhythm.

In health the heart beat is initiated in the sino-atrial node (pace-

maker) which lies near the entry of the superior vena cava into the right atrium. The impulse spreads through both atria and thence to the atrioventricular node. The AV (atrioventricular) node is continuous with the bundle of His and its branches. The commonest disorders are listed below.

Sinus tachycardia (Fig. 17)

The cardiac impulse arises normally, and the electrocardiogram is normal in form. The pulse rate is increased above 90 or 100 (adults). Sinus tachycardia may result from emotion, exercise, fever, hyperthyroidism and anaemia.

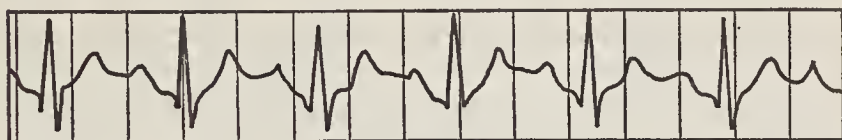


Fig. 17. Sinus tachycardia. Pulse rate 120. Timing marks on this and subsequent electrocardiograms are at 0.2 second intervals.

Sinus bradycardia (Fig. 18)

Again the electrocardiogram is normal in form, but the heart rate is less than 60/minute. Sinus bradycardia occurs in athletes, and in patients with increased intracranial pressure, myxoedema and jaundice.

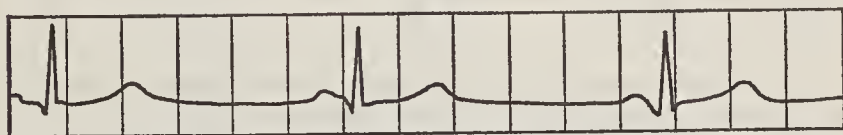


Fig. 18. Sinus bradycardia. Pulse rate 55.

Sinus arrhythmia (Fig. 19)

The cardiac impulse arises normally in the sino-atrial node, the rhythmicity of which varies; the heart rate increases with inspiration and

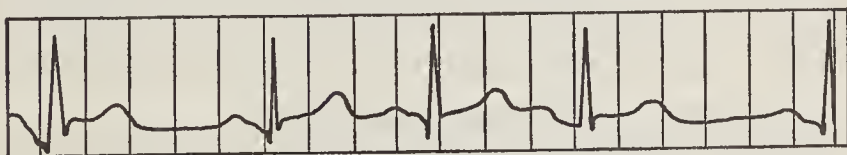


Fig. 19. Sinus arrhythmia.

diminishes with expiration. The electrocardiogram is normal apart from variation in the R-R intervals. This arrhythmia is a normal finding in young people; it is increased by deep breathing and abolished by exercise.

Extrasystoles or ectopic beats (Figs 20, 21)

These beats arise from foci in the atria or ventricles which stimulate the heart before the next sinus beat is due. In ventricular extrasystoles P waves are absent and the QRS complexes are broad, the T wave pointing in the opposite direction to the major deflection of the QRS. The extrasystole comes prematurely and is followed by a pause (the compensatory pause).

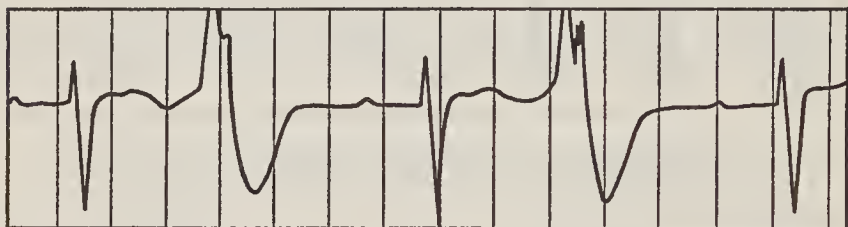


Fig. 20. Ventricular extrasystoles. Note that there is no P wave before the second extrasystole, and there is an abortive P wave just before the first extrasystole.



Fig. 21. Atrial extrasystoles. Note the abnormal (inverted) P wave. The R-R interval is longer after the extrasystole than in the normal cycle.

The electrocardiogram of an atrial extrasystole shows the P wave to be abnormal in form, but the QRS which follows it is normal. The pause which follows the extrasystole is longer than normal.

Extrasystoles are thus premature beats followed by an abnormally long pause and can be recognized by auscultation or from palpation. Extrasystoles occur both in health and in patients with heart disease. If an extrasystole follows after each normal beat as in Fig. 20 the pulse is said to be coupled (*pulsus bigeminus*). Digitalis often causes coupling.

Atrial tachycardia and atrial flutter (Figs 22, 23)

These are due to the presence of an ectopic focus in the atrium which beats regularly at a rapid rate. The P waves are abnormal in shape, but

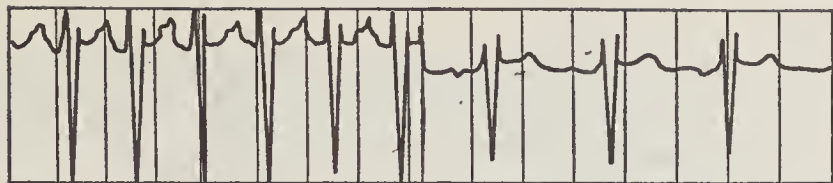


Fig. 22. Atrial tachycardia. During attack, pulse rate 225; after attack, sinus tachycardia, pulse rate 130.

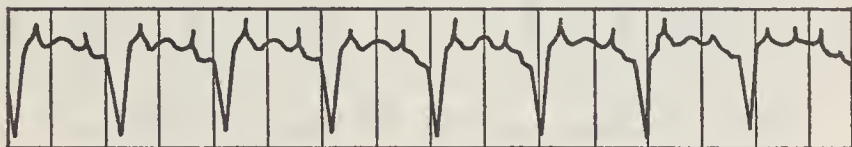


Fig. 23. Atrial flutter, 2:1 block. Atrial rate about 300. Note the two spiked flutter waves to each ventricular complex.

the QRS complexes are usually normal, although at faster rates a bundle branch block pattern (p. 125) may develop. As a rule not all atrial impulses are conducted to the ventricles. Often alternate beats are conducted when 2 : 1 atrioventricular block is said to be present. Occasionally 3 : 1 or 4 : 1 block is present and sometimes the block varies.

Atrial flutter and tachycardia may occur in hearts which are otherwise normal, in thyrotoxicosis and in rheumatic or ischaemic heart disease.

Atrial fibrillation (Fig. 24)

There is no coordinated atrial activity (either electrical or mechanical). The electrocardiogram shows *f* (fibrillation) waves representing the atrial activity instead of P waves, especially in lead V_1 . The QRS complexes are normal, but are irregularly spaced.

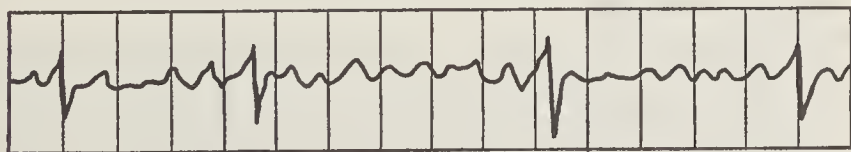


Fig. 24. Atrial fibrillation. Note the *f* waves and irregular ventricular rhythm.

Atrial fibrillation is recognized clinically by complete irregularity of the pulse both in rate and volume. Mitral valve disease, ischaemic heart disease and thyrotoxicosis are the commonest causes of fibrillation.

Heart block (Figs 25, 26)

In first degree atrioventricular block the P-R interval exceeds 0.2 second and all the impulses reach the ventricles. When some impulses fail to reach the ventricle but others do reach it, then there is second degree heart block. In complete heart block (third degree) the atria and ventricles beat independently, i.e. they are dissociated, and the ventricular rate is usually slow, 20–40/minute.

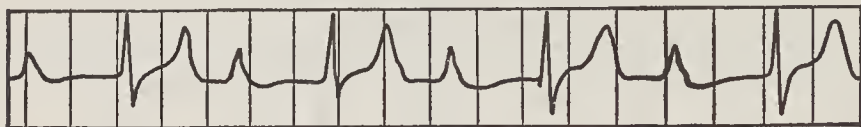


Fig. 25. First degree heart block. P-R interval = 0.42 second.

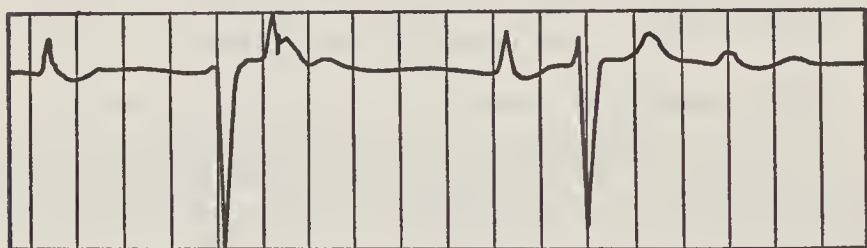


Fig. 26. Complete heart block. Atrial rate 55; ventricular rate 39.

Ventricular fibrillation

A similar mechanism operates in the ventricles as in the atria with atrial fibrillation. There are no distinct QRS complexes, but bizarre undulations of the tracing of irregular height and rate.

The electrocardiogram in some other conditions**Ventricular hypertrophy**

Ventricular hypertrophy is diagnosed principally from the chest leads and in particular V_1 and V_6 . Fig. 28 shows the appearance of the QRS complexes in V_1 (over the right ventricle) and V_6 (over the left ventricle). It must be appreciated that the QRS complex from any lead represents the algebraic sum of the electrical activity of both ventricles. At any point in time the ECG will show an R wave if the resultant is directed towards the electrode and an S wave if the resultant is going away from the electrode. Ventricular hypertrophy is associated with an increase in the electrical activity of depolarization. As a result there is an increase in the magnitude of the QRS deflections best seen in the chest leads.

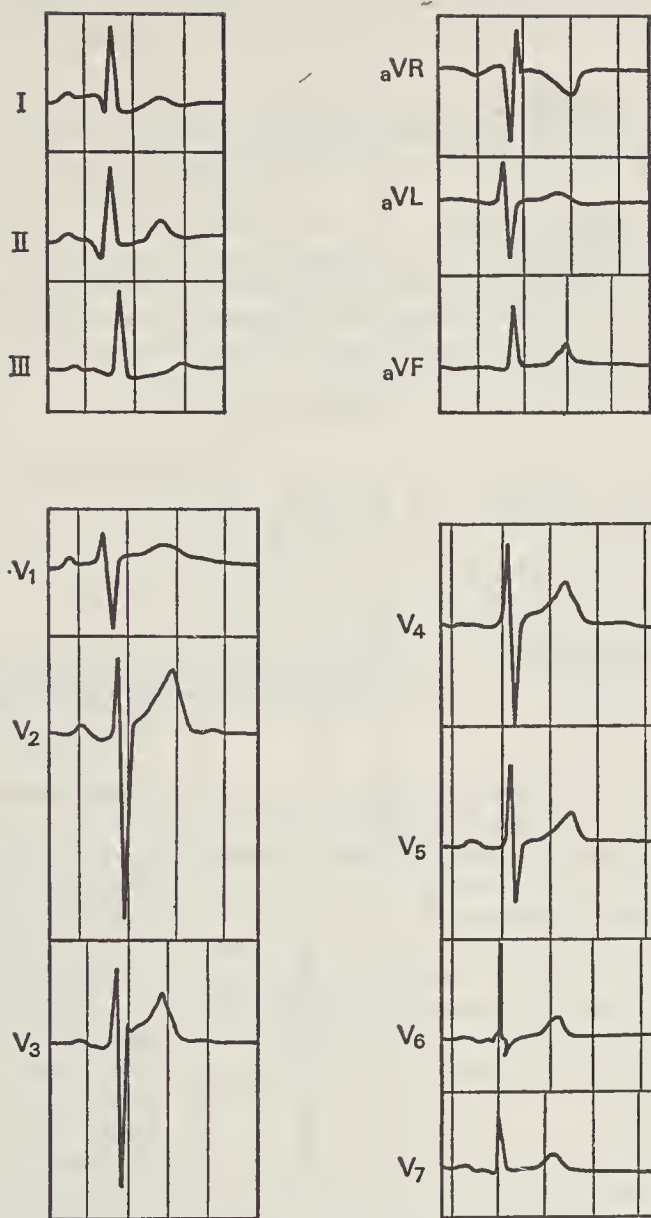


Fig. 27. The normal electrocardiogram. Standard leads (I, II, III), V (unipolar) limb leads, V (unipolar) chest leads V₁–V₇.

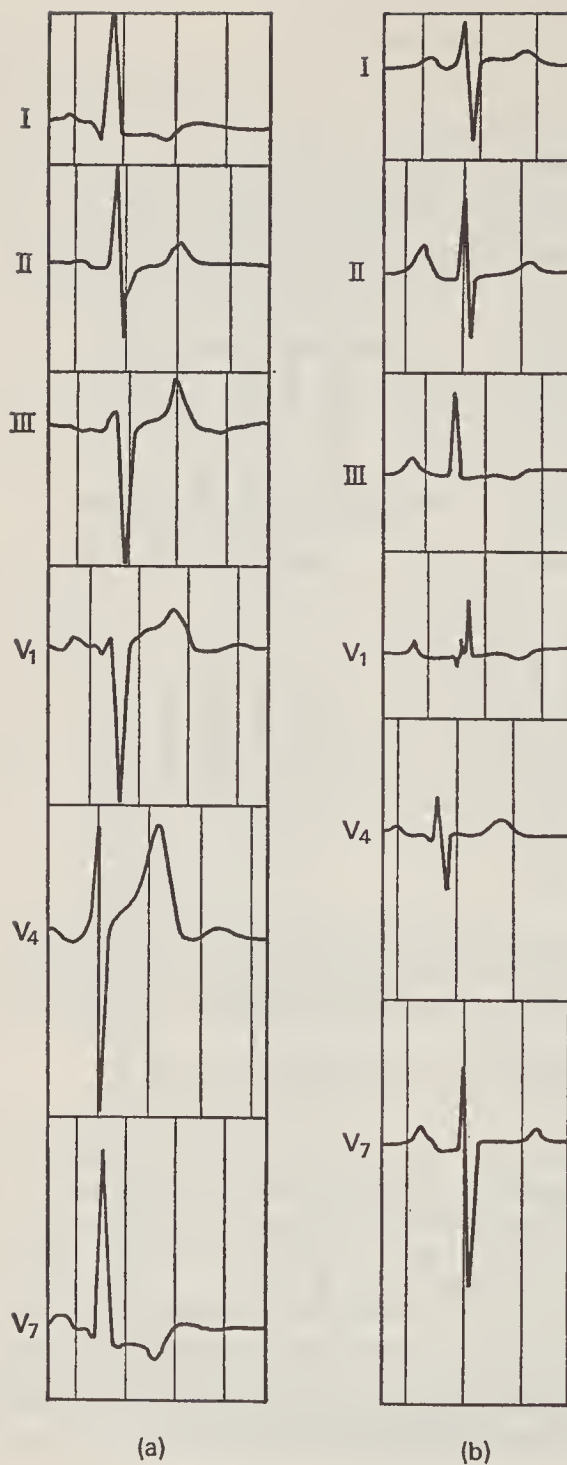


Fig. 28. (a) Left ventricular hypertrophy. Note the deep S wave in V₁ and the tall R wave in V₇. Left axis deviation, S-T depression and T wave inversion in lead I and V₇. (b) Right ventricular hypertrophy. Note the tall R wave in V₁ and deep S wave in V₇. Right axis deviation. T wave inversion in lead III and V₁. In this tracing the T wave is normal in lead II, though it is usually flat or inverted in right ventricular hypertrophy. The P wave in V₁ is sharp and peaked, suggesting right atrial hypertrophy.

Left ventricular hypertrophy (Fig. 28a). As the left ventricle lies to the left and posteriorly there is an increase in the size of the R wave in the left chest leads (V_5 – V_7) and lead I and an increase in the S wave in the right chest leads (V_1 or V_2).

Right ventricular hypertrophy (Fig. 28b). The R wave in the right chest leads is increased and there is a deeper S wave in the left chest leads and in lead I as the increased electrical activity is associated with the anteriorly placed right ventricle.

Both types of ventricular hypertrophy may in addition show a *ventricular strain pattern*. This is an abnormality of ventricular repolarization associated with hypertrophy. It causes the T wave to point away from the affected ventricle giving ST depression and T wave inversion in leads I, aVL and the left chest leads in left ventricular hypertrophy, and in leads V_1 , V_2 , II and III in right ventricular hypertrophy. Other causes of T wave changes are mentioned on p. 127.

Bundle branch block (Fig. 29)

Owing to an interruption of a branch of the bundle of His conduction to one ventricle is delayed. The QRS duration is greater than 0.1 second and the pattern is similar to ventricular hypertrophy. For example, if the left bundle is interrupted, conduction to the left ventricle is delayed and it becomes activated at a time when it is no longer opposed by the right ventricle, and therefore produces large deflections, as in hypertrophy of the left ventricle.

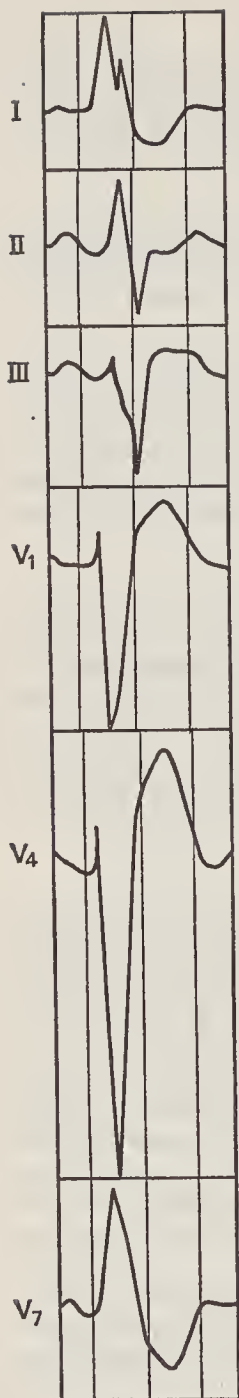
Cardiac infarction

Cardiac infarction alters the electrocardiogram by the production of abnormal Q waves or abnormalities in the S–T segments and T waves, or both. Within a few hours of infarction the S–T segments become elevated (Pardee's sign) (Fig. 30b). In a few days the T waves become inverted, often steeply so (Fig. 30c). The S–T segment gradually returns to the base line, taking several weeks to do so. T wave inversion may eventually return to normal (Fig. 30d), but some inversion usually persists. Abnormal Q waves are usually permanent. The leads showing Q waves or S–T and T wave change are determined by the site of infarct. The electrocardiograms illustrating classical anterior and inferior infarction (Fig. 31) are tracings taken several weeks after the infarction.

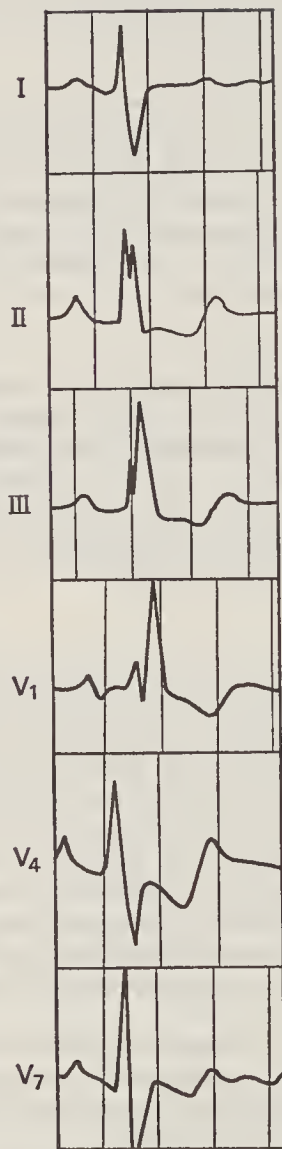
In *anterior infarction* the changes are seen in leads I and V_1 to V_4 .

In *inferior infarction* (or diaphragmatic infarction sometimes erroneously called posterior infarction) the changes are seen in leads II and III and aVF.

In *lateral infarction* the changes occur in leads I, aVL and V_5 to V_7 .



(a)



(b)

Fig. 29. (a) Left bundle branch block. Note the wide QRS complexes (0.15 seconds) and deep wide slurred R wave in V₇. (b) Right bundle branch block. Note the wide QRS complexes (0.15 seconds), rsR pattern in V₁ and deep wide S wave in V₇.

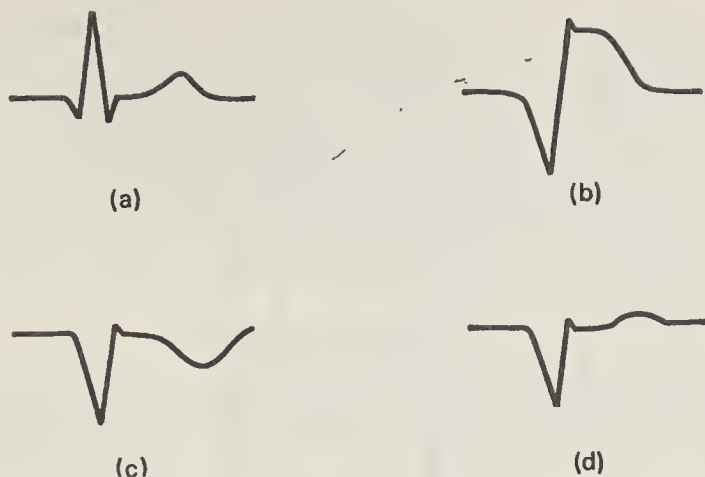


Fig. 30. The evolution of the changes in the QRS complex, S-T segment and T wave after cardiac infarction. (a) Normal pattern. (b) A few hours after infarction. A Q wave is present. The S-T segment is elevated (Pardee's sign). (c) After a time the S-T segment returns to the base line and the T wave becomes steeply inverted. (d) After a further period the T wave becomes inverted, flat and finally upright. Note that the Q wave persists.

Estimation of aspartate aminotransferase may also help in the diagnosis of cardiac infarction (p. 59). Its level increases significantly after 6–12 hours and reaches a peak of 50–150 i.u./litre within 24–48 hours, returning to normal in 4–7 days.

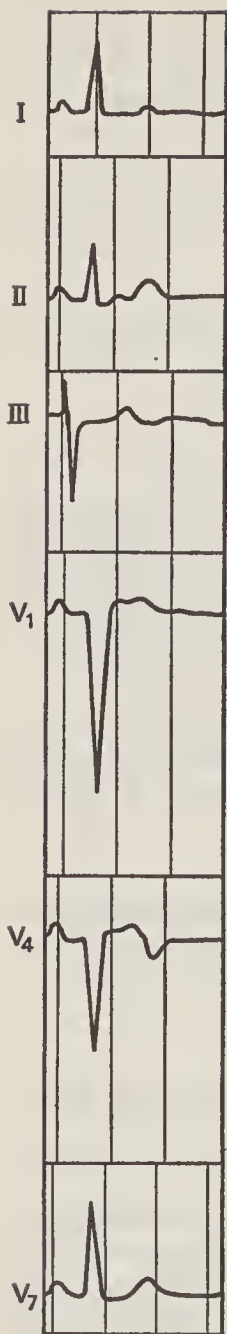
P wave changes

In right atrial hypertrophy, due for example to chronic cor pulmonale or pulmonary stenosis, the P wave is tall and sharp. In left atrial hypertrophy, especially in mitral stenosis, the P wave is broad and bifid.

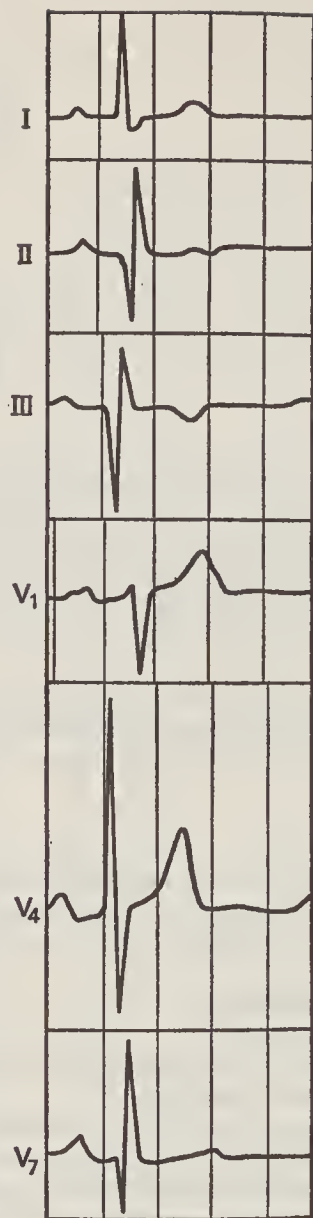
S-T segment and T wave changes

These occur in many conditions:

1. Digitalis depresses the S-T segment in all leads, especially in limb leads I and II and in the left chest leads.
2. Flat or inverted T waves occur in myxoedema and are associated with low voltage QRS complexes and bradycardia.
3. Hypokalaemia causes depression of the S-T segments and inversion of the T waves, while hyperkalaemia causes tall sharp T waves.
4. Pericarditis causes the S-T segment to be elevated in some leads, especially in lead II. The segment retains its normal concavity. This change is due to epicardial damage and is strongly suggestive of acute pericarditis. In chronic pericarditis there is T wave inversion.



(a)



(b)

Fig. 31. (a) Anterior cardiac infarction. Note the Q wave in V₁ and V₄. The T wave is low in lead I and inverted in V₄. (b) Inferior cardiac infarction. Note the Q waves in leads II and III. The T wave is slightly inverted in lead II and inverted in lead III. V₇ also shows a Q wave and a low T wave, so that this is really inferolateral infarction.

RADIOGRAPHIC EXAMINATION OF THE HEART

In addition to the standard postero-anterior X-ray of the chest much information is gained by taking more penetrated films of the postero-anterior and right lateral views.

Screening of the heart is occasionally of value in visualizing calcification in valves or in the coronary arteries, the degree of pulsation in the pulmonary arteries or aorta, and the differentiation of pulsatile from non-pulsatile shadows.

The normal cardiac outline

The heart is seen as a flask-shaped shadow, lying between the translucent lungs, about one-third of its area to the right of the midline and two-thirds to the left. The apex of the heart is internal to the mid-clavicular line.

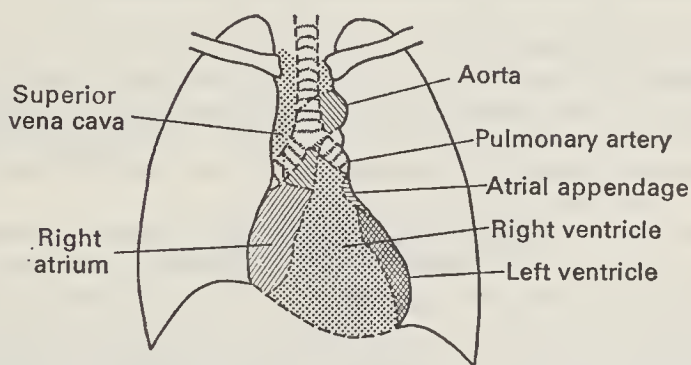


Fig. 32. The cardiac silhouette in the anteroposterior position. This is particularly useful in studying the outflow from the left ventricle, the pulmonary artery and the aortic knuckle.

The *right border* of the cardiac shadow is formed, from above downwards, by two curves:

1. A slightly curved portion, the outer edge of the superior vena cava with the ascending arch of the aorta.
2. A more convex portion, the outer border of the right atrium, which ends at the diaphragm.

The *left border* is made up from above downwards by:

1. The prominent knuckle produced by the arch of the aorta as it passes backwards, slightly to the left, then downwards.
2. The straighter line of the pulmonary artery.
3. The wide sweep of the left ventricle, ending at the apex, where it rests on the diaphragm.

In the overpenetrated postero-anterior film the left atrium can be seen, especially if enlarged, and the aorta is particularly well shown. Calcification in the pericardium or valves is usually apparent. The right lateral film is also of value in showing calcification and in addition is helpful in right ventricular hypertrophy when the cardiac shadow comes closer to the sternum and higher up the sternum than usual.

Common alterations in disease

Position of the heart in the chest

Displacement of the heart as a whole is seen in pleural effusion, pneumothorax and fibrosis of the lung. In distension of the stomach and obesity, the heart is raised with the diaphragm and the apex tilted upwards. The common type of scoliosis (convexity of the curve to the right) is a frequent cause of displacement of the heart to the left. In narrow chests the heart often lies centrally and seems small and slender.

Shape and size of heart

Prominence and undue convexity of the left ventricle is seen in aortic regurgitation, in aortic stenosis and in hypertension. In its typical form the outline may be described as boot-shaped. An enlarged left ventricle is well seen (on screening) in the left oblique view when it extends backwards to cover partly the shadow of the spine.

The left atrium is enlarged in mitral stenosis and mitral regurgitation. The left atrial appendix shows in chest X-rays as a straightening or convexity of the normally concave left border of the heart below the pulmonary artery.

The overpenetrated grid film is particularly useful in detecting left atrial enlargement. The outline of the enlarged left atrium can usually be seen through the right atrial shadow on the right border. On the left, the dark shadow of the left main bronchus is displaced upwards by the enlarged left atrium and lies more horizontally.

Left atrial enlargement can also be assessed by giving the patient barium paste to swallow and screening him in the right oblique position. The oesophagus is displaced backwards and to the right by the enlarged left atrium.

Shape and size of the aorta and superior vena cava

Enlargement of the ascending aorta occurs in syphilitic aortitis with aneurysm, in aortic stenosis (post-stenotic dilatation) and in aortic regurgitation.

Unfolding of the aorta is seen in atheroma, especially when hypertension is associated. The shadow of the superior vena cava is widened in congestive heart failure.

Pulsation of the heart and aorta

An abnormal degree of aortic pulsation is a feature in the cardioscopic examination of cases of aortic regurgitation. In mitral regurgitation, the shadow of an enlarged left atrium is seen in the anteroposterior view to expand markedly during ventricular systole. A saccular aneurysm of the aorta appears as a rounded outgrowth from some part of the aorta, which itself will often be dilated. Its relation to the course of the aorta and its pulsation help to distinguish it from an intrathoracic tumour.

The hilar shadows and lung fields

In mitral stenosis, where the left atrial pressure is significantly raised, horizontal lines (Kerley B lines) due to engorged sub-pleural lymphatics are seen at the lung bases. The hilar shadows are heavy and rather ill-defined. Similar changes are found in left ventricular failure.

When the pulmonary blood flow is greatly increased, as in atrial septal defect, the main branches of the pulmonary artery are increased in size—'*pulmonary plethora*'. This is especially obvious when some of the branches are seen end-on near the hilum. Conversely, in Fallot's tetralogy the vascular markings in the lung fields are inconspicuous—'*pulmonary oligæmia*'.

CARDIAC CATHETERIZATION AND ANGIOCARDIOGRAPHY

Catheterization of the heart is performed to obtain detailed haemodynamic information and to assist in the diagnosis or assessment of heart disease. Right heart catheterization is performed by passing a flexible radiopaque catheter via a vein into the right atrium, right ventricle and pulmonary artery under fluoroscopic control. The left heart can be entered by the retrograde passage of a catheter from an artery to the aorta and thence to the left ventricle; or by puncturing the atrial septum with a needle passed through the venous catheter and subsequently advancing the catheter over the needle into the left atrium and left ventricle. Information can be obtained in several ways:

1. The pressure within the cardiac chambers and vessels can be measured. Abnormal pressure gradients across valves may be demonstrated.

2. The cardiac output can be measured using either the Fick principle or dilution techniques.

3. Septal defects may be demonstrated by passing the catheter through the defect and by measuring the oxygen saturation of blood samples taken from different chambers. For example if the oxygen saturation in the pulmonary artery and right ventricle is 80% and in the right atrium is 65%, this would indicate a shunt of blood through a

ventricular septal defect from the left ventricle, and the amount of the shunt can be calculated.

Angiocardiology consists of the injection of contrast material into the chambers of the heart, the great vessels (aorta and pulmonary artery) or the coronary arteries, while serial X-ray films are exposed. Where the abnormality to be demonstrated is dynamic in nature, e.g. mitral regurgitation, this is best demonstrated by high speed cine-angiocardiology. Where detailed anatomy is to be shown, the serial exposure of full sized X-ray films in one or two planes, using a mechanical film changer, is preferable.

These two techniques are used together to identify cardiac lesions and assess their severity; and to indicate patients suitable for cardiac surgery.

6

ELEMENTARY HAEMATOLOGY

Examination of Stained Films — Estimation of Haemoglobin — Packed Cell Volume — Red Cell Counts — White Cell Counts — The Differential White Cell Count — Platelet Count — The Absolute Values — The Reticulocyte Count — The Coagulation, Bleeding and Prothrombin Times — Fragility of the Red Cells — The Coombs Test — Bone Marrow Puncture — Sedimentation Rate — Blood Grouping — The 'LE' Cell Phenomenon — Parasites in the Blood

EXAMINATION OF STAINED FILMS

The first step in the examination of the blood is the preparation of stained films. The examination of a properly made blood film by a competent observer, together with a determination of the level of haemoglobin (or of the volume of packed cells), is enough in the majority of cases either to exclude disease of the blood or to suggest a diagnosis. Red cell counts are tedious and inaccurate, unless electronic methods of counting are available, and such counts are seldom necessary.

Films may be made from anticoagulated (preferably with sequestrene) venous blood (p. 327). In this case they should be made as soon as possible and not from blood which has been allowed to stand. For most purposes they are best made from a drop of peripheral blood obtained by pricking the lobe of the ear or the finger (or the heel in the case of infants) with a sharp sterile needle. Blood for haemoglobin level and other purposes can be obtained at the same time.

The ear or finger should be pink and warm. The puncture should be made vertically to a depth of 2–3 mm so that a free flow of blood is obtained. Wipe away the first few drops and then take samples when the blood is flowing freely. Squeezing should be avoided. The needle used must be flamed between each patient.

The slides used for making films should be entirely free from grease. To ensure this they should be placed for 48 hours in dichromate cleaning fluid (potassium dichromate 20 g, dissolved in water 100 ml, to which are then added 900 ml of concentrated sulphuric acid). They should then be well washed in running water and stored in 95% ethanol. When required, they should be picked out with forceps, allowed to drain and then dried off with a clean cloth. They should finally be rubbed with a clean handkerchief. Slides may also be satisfactorily cleaned

by polishing them with the finest emery paper and then leaving in ethanol until required.

If slides have to be cleaned in a hurry, glacial acetic acid followed by water and alcohol gives good results.

How to make films

Apply one end of a slide to a drop of blood and place the slide on a level surface, holding it with the thumb and index finger of the left hand. The narrow edge of a second slide is placed in the drop and held there till the blood has spread across it; it is then drawn slowly over the whole length of the first slide. The inclination of the second slide to the first should be 45° , and there should be no pressure whatever between the two surfaces. The more slowly one slide is drawn over the other, the thinner is the resulting film. Smooth spreading of the film is aided by warming the first slide in the flame of a spirit lamp before applying it to the drop of blood. After the blood is spread it should be dried by being waved rapidly in the air to prevent undue shrinkage of the cells.

How to stain the film

Either of the two following methods gives excellent results.

Jenner's stain. (The stain consists of a 0.5% solution of a specially prepared crystalline compound of methylene blue and eosin in pure methyl alcohol.) Films are made in the usual way. As soon as they are dry, a few drops of the solution are poured on, and they are covered with watch-glasses to prevent evaporation and precipitation of the stain. Pour off in one to four minutes. Rinse in *distilled* water till pink (5–10 seconds). Dry rapidly high over a flame or by waving in the air. Mount in xylol balsam. In a successful film the red corpuscles are brownish-red, nuclei are blue, platelets mauve, the granules of polynuclear cells and myelocytes red, basophils dark violet, bacteria, filarial and malarial parasites blue.

Leishman's stain. This is a simplification of the method of staining first introduced by Romanowsky. The dry film is well covered with the stain, which should be evenly distributed over the entire slide or cover-glass. At the end of 1 minute, double the quantity of distilled water is carefully added and mixed with the stain. At the end of 7 minutes, the mixture is poured off and the film covered with distilled water for 2 minutes. The water is then washed off with fresh distilled water, and the film gently blotted dry with clean blotting paper. When dry it can be mounted in xylol balsam or examined directly under the oil-immersion lens.

Examination of the film

First make a general examination of the film. This is best done by placing a drop of mountant on the film, covering it with a cover-glass and using the low power (16 mm objective) followed by the high power (4 mm objective), but in practice most observers place a drop of immersion oil directly on the stained film and use the oil immersion lens, which is needed in any case for the examination of abnormal cells. Observe whether the film is properly spread. Then observe whether red cells, white cells and platelets are present in about normal proportions, or whether there is a gross excess of white cells, suggesting leukaemia (Plate IX), or a severe deficiency of platelets, suggesting thrombocytopenia. For parasites in the blood, Plate XV.

The observer should then turn his attention to the morphology of the red cells. The points to be observed are as follows:

The size of the red cells. Normal red cells form a homogeneous population with a fairly narrow variation around a mean diameter. For special purposes red cell diameter distribution curves can be constructed. Normal curves are shown in Fig. 33a. The mean diameter in health varies from 6.7 to 7.7 μm with an average of 7.2 μm . The width of the base of the curve (or more accurately the coefficient of variability) gives an indication of the degree of inequality in the size of the red cells.

In states of iron deficiency (Fig. 33c) the mean cell diameter is reduced (microcytosis) and the width of the base of the curve is somewhat increased (anisocytosis).

In anaemia due to vitamin B12 and folate deficiency the mean cell diameter is increased (*macrocytosis*) and the width of the base of the curve may be greatly increased (*great anisocytosis*) (Fig. 33b).

Variation in the shape of the red cells, usually the presence of small misshapen cells, is referred to as *poikilocytosis*. It is seen in any severe anaemia, but particularly in pernicious anaemia and haemolytic anaemias.

The staining of the red cells. Red cells *in vivo* are biconcave discs, and even when fixed and stained they are usually paler at the centre than at the periphery. Poor staining of the red cells (*hypochromia*), along with microcytosis, is characteristic of states of iron deficiency (Plate X) but is also seen in thalassaemia. In long-standing iron deficiency virtually all the cells are hypochromic, whereas with the recent development of iron deficiency, as from recent haemorrhage, a mixed population of normal cells and hypochromic microcytes is found. The presence of unduly large well stained red cells, without a pale centre, along with consider-

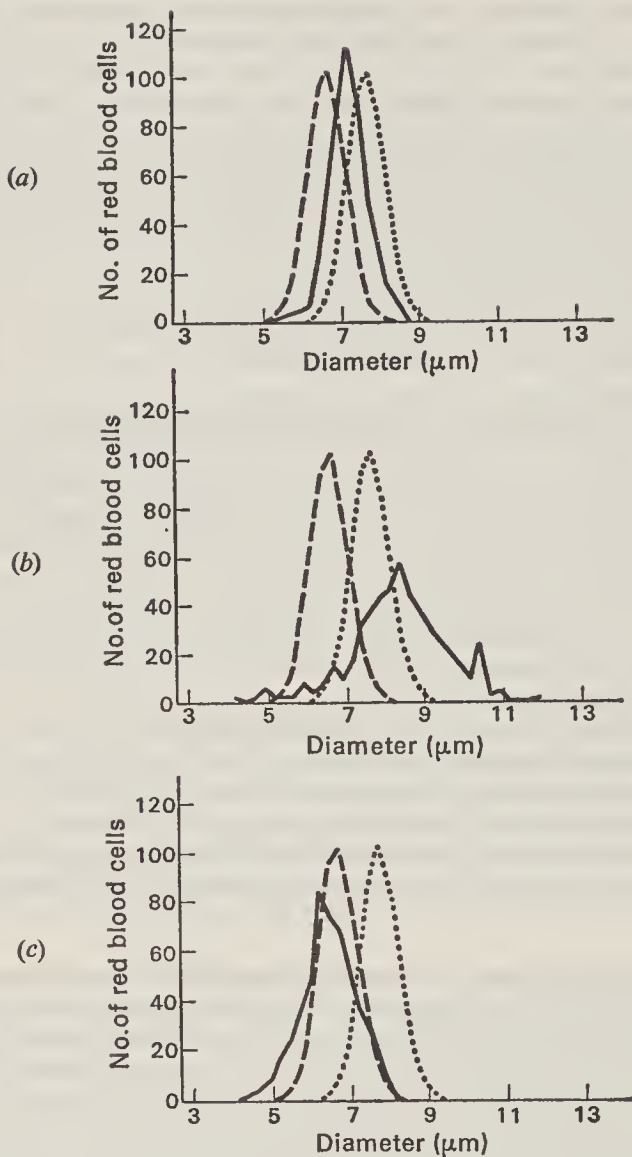


Fig. 33. Distribution curves for the diameter of the red cells. The dashed and dotted lines show the ideal curves for smallest and largest (± 3) mean diameter with normal limits. (a) A normal woman. (b) A patient with pernicious anaemia. (c) A patient with iron deficiency anaemia.

able anisocytosis and poikilocytosis is characteristic of anaemia due to vitamin B12 or folate deficiency. Increased segmentation of the nuclei of neutrophils is also present in such circumstances (Plate X). Mixed pictures of macrocytosis and microcytosis with hypochromia are not uncommon in anaemia caused by multiple nutritional deficiencies and by malabsorption.

The presence of large numbers of cells showing diffuse bluish coloration (*polychromasia*) and speckled blue discoloration (*punctate basophilia*) suggests either intense regeneration (i.e. after haemorrhage or after the administration of a needed specific haemopoietic agent) or some type of haemolytic anaemia. Marked punctate basophilia is characteristic of lead poisoning.

The presence of a number of unusually deeply-stained round cells (*spherocytes*) together with polychromatophilia and punctate basophilia is characteristic of hereditary spherocytosis (*familial acholuric jaundice*) (Plate X).

Unduly flat cells, which are the opposite of spherocytes, show colour at the periphery, a pale ring and colour again at the centre. They are described as *target cells*, and are found particularly in familial anaemias, associated with the presence of abnormal haemoglobins (*thalassaemia* and *sickle-cell anaemia*). *Sickle cells* themselves may be seen in the blood film together with target cells in sickle-cell anaemia, owing to the presence of the abnormal haemoglobin S (see Plate X).

The presence of nucleated red cells. Normoblasts can be distinguished from lymphocytes (for which they are apt to be mistaken at first glance) by (a) their more homogeneous and dense dark staining of the nucleus, and (b) their red cytoplasm.

Megaloblasts are larger cells with a characteristically stippled nucleus and polychromatophilic cytoplasm.

Nucleated red cells (usually normoblasts) may be found in any severe anaemia and in states of intense blood regeneration. They are also found in leukaemia. Megaloblasts are occasionally seen in the peripheral blood in conditions in which they are abundant in the bone marrow (pernicious and other megaloblastic anaemias), but this is an inconstant finding. The presence of a number of primitive red and primitive white cells in the peripheral blood described as *leucoerythroblastic anaemia*, and is due in most instances to infiltration of bone marrow by leukaemia, carcinoma, myeloma cells or by fibrous tissue (as in myelofibrosis).

ESTIMATION OF HAEMOGLOBIN

Haldane's and Sahli's methods of estimating haemoglobin, which depended on dilution and the matching of colour with the eye, are no

longer used in laboratories; though it is better for the clinician to make use of one or them in deciding whether a patient is anaemic or not than to have no method of estimating haemoglobin at all.

Accurate haemoglobinometry is now carried out by photocolormetric methods. The blood to be examined is diluted, usually with a solution containing potassium cyanide and potassium ferricyanide (Drabkin's agent), which converts oxyhaemoglobin, reduced haemoglobin methaemoglobin and carboxyhaemoglobin into cyanomethaemoglobin. The optical density of this solution is then compared in a photoelectric colorimeter with that of a standard solution of cyanomethaemoglobin. For details of such methods larger works must be consulted.

A modification of the MRC Grey-Wedge Photometer (marketed as 'Haemoscope' by Keeler Optical products Ltd) works on the same principle and provides a portable and less expensive method of accurate haemoglobinometry (within the range of 6–16 g of haemoglobin/100 ml).

The normal haemoglobin varies within considerable limits and differs in the two sexes and at different ages. The mean haemoglobin of healthy men may be taken as 15 g/100 ml, and the normal range from 13.5 to 18 g/ml. The mean figure for healthy women may be taken as 14 g/100 ml, and the normal range from 11.5 to 16.5 g/100 ml.

PACKED CELL VOLUME

This is determined by centrifuging anticoagulated blood in a graduated tube until the corpuscles are packed down to a constant volume. The volume of packed cells is then expressed as a percentage of the original column of blood. Venous blood may be anticoagulated with sequestrene (EDTA) or, if more accurate results are required, an ammonium-potassium oxalate mixture may be used which produces no shrinkage of cells (5 ml of blood from a vein are placed in a tube containing 4 mg of solid potassium oxalate and 6 mg of solid ammonium oxalate). With the aid of a capillary pipette, a Wintrobe's haematocrit tube (which can also be used for determining the sedimentation rate, p. 151) is then filled to the 100 mark with the anticoagulated blood and centrifuged at 2500 rpm for 30 minutes. As the original column of blood in the tube is 100 mm long, the volume of packed cells can be read directly as a percentage. This estimation is simple and accurate, and from this and the haemoglobin the mean corpuscular haemoglobin concentration (MCHC) (the most important of the absolute indices) is determined (p. 144).

RED CELL COUNTS

As already stated, this procedure is performed by visual methods is tedious, inaccurate and rarely necessary. Electronic methods of counting are becoming more widely available; but, if required, an approximate count can be made by visual methods.

The instruments necessary are a graduated mixing pipette and a counting slide. Either venous blood (p. 133) or peripheral blood (p. 327) may be employed. In the former case the bottle or tube must be well shaken before a sample is taken, and in the latter the sample must be taken from a drop of blood which flows freely without squeezing. Slowly suck up blood with the pipette till the mark 0.5 is reached. If one should go on a little beyond the 0.5 mark, the point of the pipette should be dabbed once or twice on the finger, till the blood is back at the 0.5 mark. Remove any surplus blood from the point, taking care that the column within the pipette does not move, and plunge it at once into the diluting fluid, which should be standing ready in a small wide-necked bottle. Suck up the diluting fluid as far as the mark 101. While this is being done, the pipette should be gently rotated to start the mixing. Grip the pipette firmly by its ends between the forefinger and thumb and shake thoroughly for at least 1 minute. This ensures a thorough mixing of the blood with the fluid. The column of diluting fluid which occupies the capillary part of the pipette does not enter into the mixture. Hence, if blood is sucked up to 0.5, the solution produced is in the proportion of 1 in 200. The diluting fluid in the capillary tube should now be blown out.

The counting chamber consists of a thick glass slide, with a transverse bar at its centre, the surface of which is sunk 0.1 mm below that of the slide. The bar is separated from the remainder of the slide by two transverse grooves, running parallel to it, one on each side, and is divided at its centre by a further groove, so that two preparations may be set up at the same time. The surface of the bar is ruled with two sets of squares, each smallest square having an area of $\frac{1}{400}$ (0.0025) mm². A specially ground thick cover-glass (ordinary cover-slips must not be used) is applied to the glass slide over the bar. If the slide and cover-glass are clean and properly applied, concentric colour (Newtonian) rings can be seen, when one is looking almost horizontally along the surface of the cover-glass. The space left between the under surface of the cover-glass and the upper surface of the bar is then exactly 0.1 mm in depth.

When the haemocytometer pipette has been thoroughly shaken, a few drops of the contents of the bulb are blown out and discarded. The pipette is then held at an angle of about 45° to the surface of the counting chamber, and its point applied to the narrow slit between the counting chamber and the cover-slip. The fluid runs under the cover-slip by capillary attraction. This manoeuvre requires some practice. Bubbles must be avoided and the fluid must exactly fill the space between the bar and the cover-slip. If any fluid overflows into the grooves, the counting chamber and cover-slip must be cleaned and the whole operation repeated.

When the counting chamber has been successfully filled, the preparation is set aside for 2 minutes or so, for the corpuscles to settle. It is then examined with the low power to see whether any air bubbles or foreign bodies are present

and whether the corpuscles are distributed with fair uniformity throughout the field, after which the high power (No. 2 eye-piece and 4 mm objective) is used for counting. The microscope must be vertical and should be provided with a condenser and a diaphragm. The light should be gradually cut off until the red cells become clearly visible. Under the 16 mm objective the little squares will be seen to be marked off into sets of 16 by double ruling. (Should the lines marking off the squares be only dimly seen, it may be necessary to intensify them. This is best done by rubbing the surface of the platform with a little finely powdered graphite—the scrapings from a very soft lead pencil—and then polishing it with soft chamois leather.)

At least 4 sets of 16 squares should be counted under the 4 mm objective. The squares in each set should be gone over systematically in horizontal rows of 4 at a time. Of the corpuscles which lie *upon* the lines bounding the row, only those on the upper and the left hand lines should be counted. The number of corpuscles in each of the four sets should be approximately equal.

Calculation. Count the corpuscles in each of the 4 horizontal rows from above downwards. The total is the number of corpuscles in 16 squares. Count in this way 5 sets of 16, and divide the total by 80, which gives the average of corpuscles in one square. But the dimensions of this square are:

$$\frac{1}{400} \times \frac{1}{10} = \frac{1}{4000} \text{ mm}^2$$

Therefore, if there are x corpuscles in this dimension there will be $4000x$ in $1 \mu\text{l}$. But the blood was diluted 200 times. Therefore in $1 \mu\text{l}$ of blood there will be $4000x \times 200$ corpuscles.

Suppose, for example, that one finds a total of 480 corpuscles in the 80 squares. This gives an average of 6 corpuscles per square, or 6×4000 ; i.e. $24\,000/\mu\text{l}$ of *diluted* blood, or $4\,800\,000/\mu\text{l}$ of pure blood, if the dilution was 1 in 200. Thus if a counting chamber with the Neubauer ruling (Fig. 34) is used and 5 sets of 16 small squares (or 80 small squares) are counted, the addition of four noughts to the figure for the number of red cells in 80 small squares will give the number of red cells per microlitre (μl) of blood.

The normal concentration of red cells varies within considerable limits, and in adult life it varies in the two sexes. In men the average figure is $5\,500\,000/\mu\text{l}$. The range in health is from about $4\,500\,000$ to $6\,500\,000/\mu\text{l}$. In women the average figure is $4\,800\,000/\mu\text{l}$ and the limit in health about $3\,900\,000$ to $5\,500\,000/\mu\text{l}$. As the error in visual red counts is up to $500\,000$, differences of less than this between two counts obtained by this means should be ignored; preferably all such counts should be expressed to the nearest half-million.

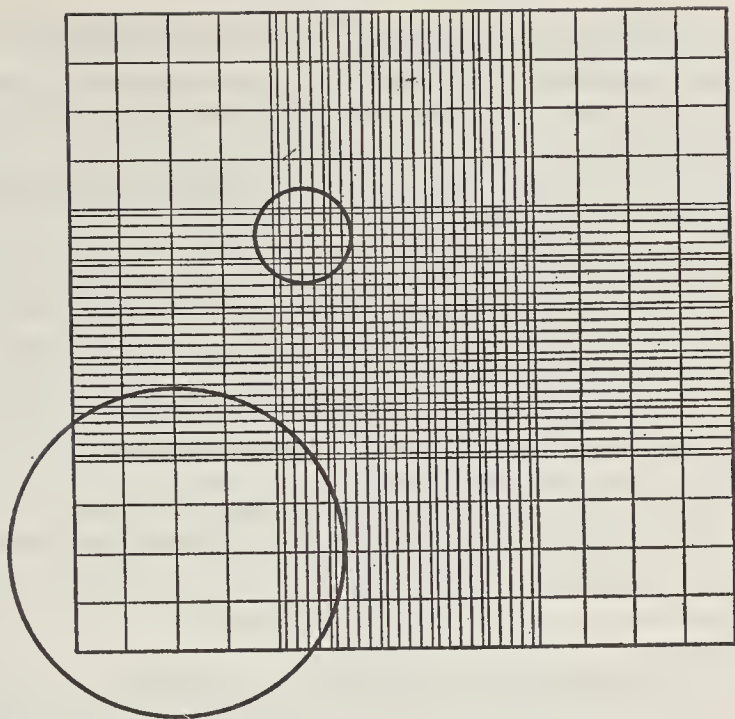


Fig. 34. A counting slide with the Neubauer ruling. The small circle shows the view seen under the 4 mm objective, used in counting red cells. The cells present in 5 sets of 16 small squares (marked off by double ruling) are counted. This number, multiplied by 10 000 gives the number of red cells per μl of blood. The large circle shows the view under the 16 mm objective used in counting white cells. The cells present in 4 large squares (made up of 16 smaller squares) at the four corners of the ruling are counted. This number multiplied by 50 gives the number of white cells per μl of blood.

WHITE CELL COUNTS

White cell counts too are now usually carried out by electronic techniques, but an approximate count, can, if necessary, be made by visual methods.

A special pipette is supplied for this purpose with the Thoma-Zeiss instrument. It is used in the same way as the red corpuscle pipette, but the blood is less dilute. The best diluting fluid is one containing 1 ml of glacial acetic acid in 100 ml of water, to which enough of a watery solution of methyl green or gentian violet has been added to give the mixture a decided colour. This mixture dissolves all the red cells while it stains the nuclei of the white.

A large drop of blood should be allowed to exude before the pipette is filled.

The blood should be sucked up to the mark 0.5, the end of the pipette wiped and diluting fluid taken up to the mark 11.

Owing to the relatively large calibre of the pipette, the blood is apt to run out of it, so the pipette should be kept in a horizontal position as soon as it is filled with blood.

The blood and fluid are mixed as already described. This produces a dilution of 1 in 20. A drop is then placed on the counting-slide with the same precautions as in the case of the red cells.

The leucocytes in the whole of the cross-ruled area of the Thoma counting chamber, which contains 400 small squares of 1 mm^2 , should be counted. Once one has learned to distinguish leucocytes from other objects, this can be done under the 16 mm objective. Several preparations should be made, and the total counts averaged, unless one has a special counting chamber (e.g. Neubauer ruling, Fig. 34) which provides several blocks of 1 mm^2 . The *calculation* is made as follows. The number of white cells counted in 1 mm^2 is that present in 0.1 mm^3 of the diluted fluid. This figure multiplied by 200 gives the white cells per microlitre (μl) of blood.

In leukaemia, where a very large excess of leucocytes is present, one can easily count the red and the white cells in the same drop. For this purpose a 3% solution of sodium citrate just coloured with gentian violet is to be preferred for diluting the blood. This stains the nuclei of the whites, and at the same time preserves the reds. The dilution and calculation are the same as for the red cells.

After use, the diluting pipettes should be thoroughly cleaned. They should be washed out (a) with distilled water, (b) with absolute alcohol, and (c) with ether. A stream of air should then be blown through till the glass ball in the chamber moves freely without tending to adhere to the sides. A few drops of antiformin sucked into the pipette will quickly disintegrate any organic matter left behind. Coagulated blood may be removed from the capillary tube with a horse-hair. If the blood adheres firmly to the pipette, it may be removed by repeated rinsing with a strong alkali or acid, or if necessary digested away with pepsin.

The *normal white cell count* is about $7000/\mu\text{l}$ in the adult. The number varies within considerable limits even in health, and counts of from 4000 to 11 000 are not necessarily abnormal. In early childhood much higher numbers are reached.

An absolute increase in leucocytes is referred to as *leucocytosis* and of lymphocytes as *lymphocytosis*. Apart from leukaemia, which will be mentioned later, important causes of *polymorphonuclear leucocytosis* are septicaemia and pyaemia, the presence of pus or an abscess anywhere in the body, any infection with pus-forming organisms, such as peritonitis, pneumonia, meningitis, and tonsillitis. *Granulocytosis* may also be found after acute haemorrhage, in amoebic hepatitis, 2 or 3 days after a coronary thrombosis, and occasionally in the presence of malignant neoplasms, particularly of the liver. An *absolute lymphocytosis*, apart from leukaemia, is a much rarer finding. It occurs particularly in glandular fever or infectious mononucleosis, and after whooping cough.

THE DIFFERENTIAL WHITE CELL COUNT

A well-spread film is essential. It is useless to attempt a differential count if the white cells are congregated in trails and clumps in the tail of the film. Given a reasonably well-spread film, the edges should be avoided and the cells running in strips the whole length of the film should be examined under either the high-power or the oil-immersion lens until 200 have been counted.

The following are the varieties of leucocytes found in normal blood, with their absolute numbers and relative proportions:

Neutrophil polymorphonuclears. Cells with multilobed nucleus and fine neutrophil or faintly eosinophil granules: 2500–7500 (40–75%).

Eosinophil polymorphonuclears. Cells with multilobed nucleus and coarse, strongly eosinophil granules: 40–440 (1–6%).

Basophil polymorphonuclears (or mast cells). Cells with very pale cytoplasm, a nucleus usually bilobed and coarse basophil granules: 0–100 (<1%).

Monocytes. Cells with a characteristic notched or kidney-shape nucleus and a slightly basophilic, faintly reticular cytoplasm: 200–800 (2–10%).

Lymphocytes with round nucleus and clear basophilic cytoplasm: 1500–3300 (20–45%).

The changes in these cells in *leucocytosis* have been mentioned (p. 142).

In the lymphatic form of leukaemia there is an enormous increase in the number of the lymphocytes (Plate IX).

In the chronic myeloid form of the disease the neutrophils, eosinophils and basophils are all increased, and in addition bone marrow cells—myelocytes—appear in the blood. These are often of large size, with a single round nucleus, and contain granules which may be either neutrophilic or eosinophilic in reaction (Plate IX).

A relative diminution of the leucocytes is spoken of as *leucopenia*. As a rule in leucopenia the diminution affects chiefly the granulocytes, hence the name *granulocytopenia*. There is, of course, a relative lymphocytosis. Leucopenia occurs in:

1. Infections such as typhoid fever, undulant fever and measles.
2. Defective bone marrow function, as in aplastic anaemia or pernicious anaemia.
3. Crowding of normal cell production by malignant cells, as in leukaemia, or by fibrous tissue, as in myelofibrosis.

4. Sensitivity to many drugs, e.g. sulphonamides, thiouracil, amidopyrine, phenylbutazone, chloramphenicol.

PLATELET COUNTS

The platelets or thrombocytes are elliptical or circular bodies with basophilic cytoplasm and azurophil granules. There is approximately 1 platelet to every 20 red cells in normal conditions. An accurate platelet count may be performed by using an electronic particle counter, or by indirect phase-contrast microscopy on a Neubauer counting chamber, using a diluting fluid containing urea, which haemolyses red cells. An approximate platelet count can be performed as follows:

The skin of the ear is cleaned with ether and on the clean surface is deposited a large drop of diluent (3% sodium citrate in normal saline). The skin is then stabbed with a sharp sterile needle so that the blood oozes directly into the diluent. This prevents clumping of the platelets. With a platinum loop of 3 mm diameter some of the diluted blood is transferred to a slide and carefully covered with a cover-slip. The amount taken should be sufficient to spread out evenly between the slide and cover-slip without causing the latter to float. The preparation is ringed with petroleum jelly. Using a microscope fitted with moving stage, squared eyepiece, and 2 mm oil-immersion objective, the number of platelets and red cells is counted in several fields, thus determining the ratio of platelets to red cells. Knowing the red cell count the actual number of platelets per μl is calculated.

The normal platelet count is from 150 000 to 400 000/ μl . However, technical factors such as the development of small clots in the blood sample may give misleading low counts, and an unexpected low count should always be repeated. Figures below 150 000 certainly constitute *thrombocytopenia*. It may be found in many conditions, including idiopathic thrombocytopenic purpura, aplastic anaemia, leukaemia, pernicious anaemia and drug reactions. It may be associated with bleeding into the skin and mucous membranes. A real increase in platelets, with counts persistently above 450 000 constitutes *thrombocythaemia*. It may be seen in polycythaemia vera and after splenectomy, and may be accompanied by a tendency to intravascular thrombosis, though an excess of platelets in polycythaemia vera is sometimes associated with a tendency to haemorrhage.

THE ABSOLUTE VALUES

Mean corpuscular haemoglobin concentration (MCHC)

This is the most important of the absolute values, as it may be determined with great accuracy and will demonstrate the commonest

haematological abnormality—defective haemoglobin production due to iron deficiency.

$$\text{MCHC}\% = \frac{\text{Hb (g/100 ml of blood)}}{\text{Volume of packed red cells in } \mu\text{l/100 } \mu\text{l of blood}} \times 100$$

The normal MCHC is 30–35%. It cannot increase above 35% as the red cell stroma cannot hold a greater than normal concentration of haemoglobin, but reduction below 30%, in the great majority of cases, may be regarded as an indication for iron therapy.

Mean corpuscular volume (MCV)

$$\text{MCV } (\mu\text{m}^3) = \frac{\text{Volume of packed cells (ml/1000 ml of blood)}}{\text{Red cells (millions}/\mu\text{l)}}$$

This value depends on the red cell count and hence is less accurate than the MCHC unless electronic methods of red cell counting are available. The normal MCV is 76–96 μm^3 . The value is raised in macrocytic anaemia and reduced in hypochromic microcytic anaemia.

Mean corpuscular haemoglobin (MCH)

$$\text{MCH (pg)} = \frac{\text{Hb (g/1000 ml of blood)}}{\text{Red cells (millions}/\mu\text{l)}}$$

Like the above, this also depends on the red cell count and hence is of limited value without electronic methods of red cell counting. The normal MCH is 27–32 pg. It is usually raised in macrocytic anaemia and reduced in hypochromic anaemia.

THE RETICULOCYTE COUNT

The reticulocytes are the youngest cells in the blood. By supravital staining, that is by the application to fresh blood of a special dye before the use of a fixative, they are seen as cells slightly larger in diameter than a mature red cell, containing a delicate basophil cytoplasmic network, which disappears later as the cell matures. The normal reticulocyte count is from 0.2 to 2.0%. The count is increased in states of active regeneration, i.e. after haemorrhage and after a specific haemopoietic remedy has been administered in a deficiency state and also in haemolytic conditions.

The best dye to use for supravital staining of the blood is brilliant cresyl blue. Saturate 0.85% sodium chloride solution with the dye, filter through a double filter paper and centrifuge. Pour off the supernatant dye solution and keep it in a stock bottle. For use dilute a small quantity of 4 volumes with 2%

sodium citrate in physiological saline. Puncture the ear and draw a large drop of blood into a Wright's pipette. Follow up the blood by an equal volume of dye. Blow out on to a slide, mix thoroughly, take up again into the Wright's pipette, seal off and incubate at body temperature for 20 minutes. Then make films by the same technique as for blood films and stain them with Jenner or Leishman stain in the usual manner. The reticulocytes present among at least 500 red cells should be counted.

THE COAGULATION, BLEEDING AND PROTHROMBIN TIMES

The coagulation time of whole blood *in vitro* may be determined in capillary blood by the method of Dale and Laidlaw, or more satisfactorily in venous blood by the method of Lee and White.

In the method of Dale and Laidlaw, capillary blood obtained without squeezing from a free-flowing puncture is run into a piece of capillary tubing 1.5–2.0 mm in diameter and 15 mm long, with slightly contracted ends. The capillary bulb contains a small movable lead shot. It is immersed immediately in a water bath at 37°C and tilted up and down while the movement of the lead shot is observed. The coagulation time is the time which elapses between the moment of puncture and the moment when the shot ceases to move. The normal is up to 3 minutes but varies with the technique used. This method is simple but relatively insensitive.

In the method of Lee and White, venous blood is run into 4 un-siliconed tubes of standard size maintained at a temperature of 37°C. The coagulation time is the time from the moment of puncture to the average time at which the tubes can be tilted to an angle greater than 90° without spilling the blood. (For details of the method larger works must be consulted.) The normal coagulation time by this method is from 5 to 11 minutes (usually 6–9 minutes).

The coagulation time is increased in haemophilia, but is normal in thrombocytopenic purpura. It is also increased during the administration of anticoagulants.

The bleeding time (Duke's method) can be determined by stabbing the ear with a sharp sterile needle and removing the resulting drop of blood with a filter paper (avoiding touching the ear) every 30 seconds until bleeding ceases. The blots are recorded in series and subsequently counted. The normal bleeding time is 0–5 minutes. It is increased in thrombocytopenic purpura, but normal in haemophilia. Ivy's method, which makes use of a sphygmomanometer cuff and several punctures on the forearm, is a little more complicated, but is thought to be more reliable (normal time 7–11 minutes).

The prothrombin time. The concentration of prothrombin in the plasma is estimated by measuring the length of time taken for plasma to clot in the presence of an excess of thrombokinase and of calcium ions. Blood is withdrawn into an oxalate solution, which prevents it clotting by removing free calcium ions, and centrifuged. To the plasma, thromboplastin—Russell viper venom or an extract of brain tissue—is added to provide an excess of thrombokinase. Calcium chloride is then added and the time taken for the plasma to clot measured with a stopwatch. This is normally 10–14 seconds. Several determinations on normal subjects are made at the same time. The *prothrombin ratio* is expressed as the ratio of observed clotting time after recalcification in the specimen tested to that of normal plasma samples. The prothrombin index is the inverse of this ratio $\times 100$ and various arbitrary percentage scales are in use, leading to considerable confusion. The prothrombin ratio is increased in vitamin K deficiency in the newborn, in malabsorption, in liver disease or during the administration of anticoagulant drugs which interfere with the synthesis of prothrombin and its co-factors.

FRAGILITY OF THE RED CELLS

The fragility of red blood corpuscles is measured by their ability to resist haemolysis in diminishing strengths of salt solution. Normal salt solution is usually taken at 0.85% of NaCl, but normal red cells do not haemolyse until a dilution of about 0.5% is reached. In most forms of chronic jaundice the red cells are less fragile than normal cells and do not haemolyse in a salt solution appreciably less than 0.4%. In acholuric jaundice the fragility of the red cells is greatly increased, and haemolysis takes place in strengths of salt approaching that of normal saline. Not uncommonly haemolysis begins at 0.6% of salt or even higher. The undue fragility of the red cells is the most constant and characteristic sign of this disorder and such wide deviation from the normal is found in no other condition.

The estimation of the fragility of the red blood corpuscles is simple. Two series of test tubes each containing about 5 ml of a range of dilutions of NaCl in water are put up, and to each of one series added a drop of blood from the patient under investigation and a drop of normal blood to each of the other series. It is not necessary to wash and centrifuge the red cells; the whole blood may be taken. The normal control series should never be omitted. The range of dilutions should extend from 0.8 to 0.2% and there should be a difference of not less than 0.05% between dilutions. The solutions can be rapidly made by placing in one burette distilled water, and in a second burette a 1% solution of NaCl in water. Into the first tube run 4 ml of the salt solution and 1 ml of water, giving 0.8% NaCl; into the following test tube put 0.5 ml less of salt solution and 0.5 ml more of water, and so on until a strength of 0.2% NaCl is reached. Each tube should be thoroughly shaken

after the dilution is made, and again after the blood has been added. Finally, the tubes are allowed to stand at room temperature until all the intact corpuscles have settled to the bottom. The first tube showing haemolysis is recognized by the faintly pinkish colour of the supernatant fluid.

This test can be performed quantitatively, the amount of haemolysis in different strengths of saline being determined by measuring the depth of colour of the supernatant fluid by means of a colorimeter. Percentage haemolysis can then be plotted against strength of saline (Fig. 35). This shows that in normal subjects the fragility curve has an

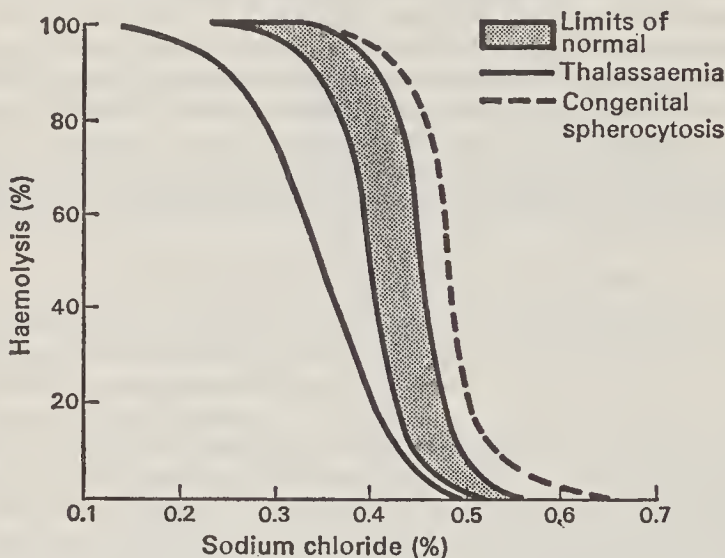


Fig. 35. Quantitative fragility curves showing increased fragility (congenital spherocytosis) and diminished fragility (thalassaemia). In general increased fragility indicates spherocytosis and diminished fragility suggests a haemoglobinopathy.

almost symmetrical sigmoid shape. Increased fragility (corresponding to increased spherocytosis) is found in familial acholuric jaundice (and only occasionally in acquired haemolytic anaemias), while decreased fragility is found in a number of anaemias, now known to be associated with the presence of abnormal haemoglobins. These are found in various parts of the world and have a particular geographical distribution. The best-known are thalassaemia (Cooley's anaemia) in persons of Mediterranean stock, and sickle-cell anaemia in persons of negroid stock. In this latter condition the red cells are normal in appearance in fresh blood but 'sickle' or assume a more filamentous shape when incubated under a sealed cover-slip at a temperature of 37°C.

In summary, an increased fragility strongly suggests congenital spherocytosis (familial acholuric jaundice), while a diminished fragility suggests an anaemia associated with an abnormal haemoglobin.

THE COOMBS TEST

Haemolysis in familial acholuric jaundice (congenital spherocytosis) is due to a congenital defect of the red cells. It is now known that in most cases of acquired haemolytic anaemia the haemolysis is due to the presence in the blood of antibodies directed against the patient's own red cells. The presence of these autoimmune antibodies can be demonstrated by means of the Coombs test, which detects 'incomplete' antibodies; that is, antibodies absorbed on the surface of red cells, which are 'incomplete' in the sense that they do not cause agglutination of haemolysis when the cells are mixed with saline dilutions of the patient's own serum. The test depends on the fact that such cells, however, will agglutinate in the presence of an anti-human-globulin serum, prepared by injecting a rabbit on several occasions with normal human serum. This anti-human-globulin serum contains a globulin which draws together the 'incomplete' antibodies in the patient's red cells, and so agglutination occurs.

In the direct Coombs test the patient's red cells are thoroughly washed in normal saline at 37°C. One drop of a suspension of these cells in saline is then mixed with a drop of a potent anti-globulin serum. (At the same time a drop of the patient's red cell suspension is mixed with a drop of saline; normal insensitized cells and cells known to be sensitized are also mixed with the anti-globulin serum—all as control observations.) The drops are gently rocked for 5–7 minutes and then examined for agglutination.

This test is better performed in a quantitative manner with different concentrations of anti-globulin serum, but the principle remains the same.

A positive direct test demonstrates the presence of autoimmune antibodies and suggests acquired haemolytic anaemia, but both 'false negative' and 'false positive' reactions can occur; the latter particularly in disseminated lupus erythematosus, rheumatoid arthritis, leukaemia and aplastic anaemia.

The indirect Coombs test is used to detect the presence of free auto-antibodies in the sera of patients with acquired haemolytic anaemia. The technique is complicated, but the principle is that washed normal group O red cells are incubated at different temperatures with the patient's serum for 2 hours. (As a control normal serum and patient's serum inactivated by heat are also used.) If the patient's serum contains auto-antibodies against red cells, these are absorbed on to the surface

of the normal red cells, which are then 'covered' with an incomplete antibody, as explained in the previous section. These red cells are then washed three times in saline and mixed with a potent anti-globulin serum as in the direct Coombs test already described. They are examined for agglutination after 5–7 minutes.

If antibody is detected by this test, a series of further tests can be performed to determine the characteristics and specificity of the antibody present, but these are matters for specialists in the field.

The indirect Coombs test can also be used for the detection of Rh antibodies (p. 153) in the sera of antenatal patients.

BONE MARROW PUNCTURE

Bone marrow puncture is employed to examine the marrow in anaemias and leukaemias, to detect certain parasites, e.g. Leishman–Donovan bodies in kala-azar, and in the diagnosis of carcinomatosis, myelomatosis and disorders of lipid storage such as Gaucher's disease. For most purposes the examination of smears of marrow aspirated through a needle is quite adequate; these can be obtained either from the sternum or from the iliac crest. For certain purposes a trephine biopsy should be taken (usually by means of a Sacker–Nordin needle) for histological section. This is essential for an accurate diagnosis of the degree of hypoplasia present in aplastic anaemias, and to establish the diagnosis in rare cases of osteosclerosis with anaemia.

The apparatus used for aspiration is a stout trochar and cannula, fitted with an adjustable stop, and so made that a hypodermic syringe fits into the top of the cannula. The skin and tissues down to the periosteum of the sternum are anaesthetized with a local anaesthetic, such as 2% lignocaine. A small incision is made in the skin with a tenotome, slightly lateral to the mid-point of the manubrium or over the middle of the sternum opposite the second or third intercostal space. The stop of the trochar is unscrewed and adjusted according to the build of the patient; the adjustment may well be 1 cm at first. The corticalis of the sternum is pierced with a boring motion, the stop being unscrewed to lengthen the projecting portion of the trochar if necessary. Some force may be required, but it is easy to recognize the entry of the tip of the trochar into the marrow cavity. When the cavity of the sternum has been entered, the trochar is withdrawn, the hypodermic syringe inserted and as few drops as possible of red fluid aspirated. Without disconnecting the syringe, the cannula is removed and a swab put over the incision. The plunger of the syringe is pushed, and as quickly as possible films are made on slides from the fluid expressed from the cannula. If marrow has been successfully aspirated, the films will contain fat and appear messy and uneven. They may be stained with Jenner's or Leishman's stain.

For the interpretation of marrow films, larger works must be consulted. Photomicrographs of normoblastic and megaloblastic marrows are shown on Plate IX.

SEDIMENTATION RATE

In health, the red cells of the blood agglutinate very little on standing; the resultant clumps of corpuscles are small and sediment slowly. On the other hand, in certain diseases the red cells agglutinate into larger clumps, which sediment more rapidly. As a sick person improves, the clumps become smaller and the sedimentation rate slower. There is no standard method for estimating the erythrocyte sedimentation rate.

The Westergren method is simplest, and the one most generally in use. 0.02 ml of 3.8% sodium citrate is sucked into a 1 ml syringe and blood is then drawn by venepuncture up to the 1 ml mark. The mixture is shaken so that it is evenly mixed. The blood is then sucked into a pipette 2.5 mm in diameter and graduated to 200 mm. This is fixed in a stand and sealed by being clipped on to a cork at the bottom. The distance settled in millimetres in one hour is the sedimentation rate. The normal is 3–7 mm, the slightly abnormal range is 8–15 mm and the grossly abnormal 15–110 mm or even higher.

In the Wintrobe method a haematocrit tube (as used for determining the volume of packed cells, p. 138) is filled to the 100 mark with oxalated blood and allowed to stand vertically for one hour. By this method the normal sedimentation for men after one hour is 0–9 mm and for women 0–20 mm. The advantage of this method is that the haematocrit tube can subsequently be centrifuged to determine the volume of packed cells and the sedimentation rate corrected for the degree of anaemia present. This can be done by means of a chart specially prepared for the purpose.

The sedimentation rate is reduced in polycythaemia and in congestive heart failure; and increased in anaemia, in all but the mildest toxic and infective conditions, and in many cases of cancer, especially in carcinomatosis. It is also increased in pregnancy, normal or abnormal. It rarely has any specific diagnostic value, but indicates the presence of a disease process of some sort, which may be anything from a cold to a carcinoma. It is excessively high in carcinomatosis, multiple myeloma and disseminated lupus erythematosus. It is of some use in distinguishing between organic and non-organic disorders, for in the presence of a raised sedimentation rate a diagnosis of a non-organic disorder should never be made until all possible investigations to exclude an organic cause have been undertaken. Conversely, a low sedimentation rate does *not* necessarily exclude organic disease.

The sedimentation rate is also useful in following the progress of patients suffering from chronic diseases. It is especially useful in chronic pulmonary tuberculosis, rheumatoid arthritis and acute

rheumatism. As long as the sedimentation rate is raised, there is an active disease process and this is frequently found a considerable time after the temperature has returned to normal. It is convenient to estimate the sedimentation rate at weekly intervals. A trend towards acceleration of the rate is an indication that the disease process is active, whereas a trend towards slowing of the rate indicates that it is subsiding.

BLOOD GROUPING

Agglutination is due to the interaction of an agglutinin (or antigen) present in red cells with an agglutinin (or antibody) present in plasma. Many different agglutinogens are present in human red cells, but in blood transfusion the important ones are the A and B agglutinogens of Landsteiner, since these are the only ones for which the corresponding agglutinins α (anti-A) and β (anti-B) are normally present in human plasma. It is evident that a person's plasma cannot contain the agglutinin for an agglutinin present in his own red cells, but with rare exceptions α or β (or both) agglutinins are present in the plasma, wherever the red cells do not contain the corresponding agglutinin. On the basis of the agglutinogens present in their red cells, human beings are divided into four groups—AB, A, B and O.

The agglutinogens and agglutinins present in the four groups together with the approximate percentage of individuals found in each group in England, are shown in the following table:

<i>Group</i>	<i>Corpuscles</i>	<i>Plasma</i>	<i>Percentage of individuals in groups</i>
AB	Agglutinogens AB	Agglutinin o	3
A	Agglutinin A	Agglutinin β	42
B	Agglutinin B	Agglutinin α	9
O	Agglutinin O	Agglutinins $\alpha\beta$	46

When a transfusion is given the danger is that the agglutinins of the recipient's plasma will agglutinate the donor's red cells. How this may come about is shown in the table opposite, where + indicates agglutination and — no agglutination.

From this table it will be seen that the group which any blood belongs to can be determined by means of a supply of suitable group A and B sera. The corpuscles of the blood under test will be agglutinated by both sera if it is of group AB, by group B serum only if it is of group A, by group A serum only if it is of group B and by neither serum if it is of group O.

<i>Recipient's group and agglutinins present in plasma</i>	<i>Donor's group and agglutinin present in red cells</i>			
	AB	A	B	O
AB Agglutinin $\alpha\beta$	—	—	—	—
A Agglutinin β	+	—	+	—
B Agglutinin α	+	+	—	—
O Agglutinin $\alpha\beta$	+	+	+	—

From this table it will also appear that blood may be given safely to any recipient from a donor of the same (or homologous) group or to any recipient from a donor of Group O (the so-called 'universal donor').

This is true in the great majority of cases, but neglects several complications, which occasionally give rise to severe and even fatal transfusion reactions in apparently compatible transfusions.

Groups A_1 , A_2 , A_1B and A_2B . Group A is strictly divisible into A_1 , accounting for four-fifths of group A persons, and A_2 , accounting for one-fifth. Group AB is similarly divisible into A_1B and A_2B . The practical importance of this fact is that A_2 cells react more weakly than A_1 cells with group B testing sera. Hence, if a low titre testing serum is used, group A_2 may be mistaken for group O and group A_2B for B. This will be avoided if high titre B serum is used, and if grouping is carefully performed.

Anti-O agglutinin is present spontaneously but rarely in A_1 , B and A_1B sera. It reacts with all O cells and also with 95% of A_2 cells, for which reason it is often called ' α_2 agglutinin'.

The extra agglutinins associated with these subgroups are usually present in small amounts, unless stimulated by a previous transfusion. Hence a single transfusion of group O blood to recipients of other groups is usually quite safe and, in practice, reactions due to repeated transfusions are rare. One danger of repeated transfusions of O blood to recipients of other groups is, however, that the formation of anti-O agglutinins may be stimulated.

The Rh factor. If red cells from a rhesus monkey are transfused into a rabbit, the rabbit's serum will subsequently cause agglutination in 85% of human bloods and no agglutination in the remaining 15%: 85% of human bloods are therefore described as rhesus positive, in that they have an Rh agglutinin in their red cells, and 15% as rhesus negative, in that they have no such agglutinin. Under normal circum-

stances Rh-negative persons have no Rh-agglutinin in their plasma, but the formation of this agglutinin may be stimulated either by a transfusion of Rh-positive blood or, in the case of Rh-negative women, by the presence of an Rh-positive fetus in utero, in some cases in which the father is Rh-positive. As mentioned (p. 150), the presence of Rh-antibodies in the serum can be demonstrated by the indirect Coombs test. This is an important measure in Rh-negative women, and can be used to forecast the occurrence of haemolytic anaemia of the newborn. If Rh-positive blood is given to an Rh-negative person, in whom the formation of Rh-agglutinins has been stimulated in one of these ways, a severe or even fatal transfusion reaction may be produced. It follows that transfusion reactions in repeated transfusions may be due to this cause, rather than to the incompatibilities mentioned above, and that Rh-negative pregnant and puerperal women can only safely be transfused with Rh-negative blood of correct A, B, O group.

Methods

Blood-grouping. If a transfusion is to be given, enough blood should be collected from the recipient to provide a sample of red cells for blood grouping and a sample of serum for cross-matching with the donor's red cells.

The recipient's finger or ear is pricked and one drop of blood allowed to drop into a small tube containing 1 ml of a 3% sodium citrate solution. This provides a suspension of red cells for grouping. Blood from the same puncture is then allowed to run into a piece of capillary tubing about 1 mm in diameter and 5 cm long. One end of this tubing is sealed and the tube then centrifuged to separate the serum, or simply left standing, when enough serum for cross-matching purposes will usually separate out. As an alternative blood may be taken by venepuncture and centrifuged. A small portion of clot is then used as the source of red cells, and the serum is used for cross-matching.

Tube method. Into two small tubes about 5 by 0.5 cm, clearly marked A and B, place respectively one volume of group A typing serum and one volume of group B typing serum, with the aid of capillary pipettes, making sure that the same pipette is never used for both sera. One volume of saline is added to each tube and then one volume of the red cell suspension prepared as above. The tubes are inverted to ensure thorough mixing and then left at room temperature for 2 hours. They are then gently flicked with the finger, when the presence or absence of agglutination is usually quite obvious. If there is any doubt, the contents of the tube can be taken up in a Pasteur pipette, transferred to a slide, and examined under the 16 mm objective. Group AB cells are agglutinated by both A and B sera. Group A cells are agglutinated by group

B serum, but not by group A serum. Group B cells are agglutinated by group A serum, but not by group B serum, and group O cells are not agglutinated by either group A or group B-sera.

Tile method. This is less accurate than the tube method, but requires less apparatus. It should only be used in emergency and by persons with experience of blood grouping.

Large drops of group A and group B typing sera are placed on a white porcelain tile and clearly marked A and B. To each, a very small drop of the patient's whole blood is added and the tile gently rocked. Usually agglutination is clearly visible to the naked eye before the fluid dries on the tile.

Cross-matching. This must never be omitted. The simplest method is to place one volume of the recipient's serum (removed by means of a fine pipette from the capillary tube prepared as above), one volume of saline and one volume of a 5% suspension of the donor's blood in 3% citrate (obtained as described under blood grouping) in a small tube. The tube is then inverted, left at room temperature for 2 hours and examined for agglutination exactly as described under blood grouping. In emergency a large drop of the patient's serum may be mixed on a slide with a drop of a 5% suspension of the donor's red cells obtained as above. The slide should then be left for 10 minutes with occasional rocking and examined under the 16 mm objective.

More satisfactory cross-matching may be achieved by carrying out the Coombs test, using the donor cells and the recipient serum. Many modifications have been devised to render cross-matching more sensitive, and special reference books should be consulted. Rare antigenic blood groups, other than the ABO and Rh systems, may give rise to difficulty; but a carefully conducted cross-match of cells as above should avoid serious transfusion reactions. If repeated incompatibility is found on the cross-match, assistance from a specialist centre should be sought.

THE 'LE' CELL PHENOMENON

If anticoagulated or defibrinated blood from a patient with disseminated lupus erythematosus (DLE) is incubated *in vitro* for 2 hours, the nuclei of some polymorphonuclear leucocytes are lysed and taken up by other polymorphonuclear leucocytes. The nuclei appear as large spherical bodies staining pale purple in the cytoplasm of the ingesting cell, which is then known as an LE cell. Such cells are not found in fresh blood or bone marrow from patients with DLE, but only appear after incubation *in vitro*. If repeated examinations are made, they are found in a high proportion of patients suffering from DLE and they are not found in patients suffering from other diseases.

Two methods are in general use. The patient's defibrinated blood

may be incubated for 2 hours at 37°C. The blood is then centrifuged and films made from the buffy coat are stained with Jenner's or Leishman's stain. Alternatively the patient's serum is incubated with a suspension of normal leucocytes, when the same phenomenon may be observed.

PARASITES IN THE BLOOD

These may be looked for in fresh blood under a coverslip, in which some of them, e.g. microfilariae and trypanosomes, may be seen alive and moving; or in fixed and stained films, which may be thick or thin. The making of thin films has already been described (p. 134).

Fresh films. The slides and cover-slips must be clean and free from grease, and should be held by the edges so that the fingers do not touch their surfaces. Prick the finger or lobe of the ear and wipe away the first drop of blood. When a second has accumulated, just touch it with the centre of a cover-slip and lower the latter carefully on to the centre of a slide. The blood spreads out into a film. If a drop of blood of the right size has been used the centre of this film is almost colourless. If the centre is red, apply a little pressure. The film should be examined, with the light well cut down, in turn under the 16 mm, 4 mm, and, if necessary, 2 mm objectives.

Thick films. The thick film method enables a much larger amount of blood to be examined in a given time than is possible with thin films and is therefore valuable for the detection of parasites when these are present in small numbers. Take a drop of blood on the centre of a slide and spread it with a triangular needle until print can be clearly seen through it while it is still wet. Allow the film to dry thoroughly, by leaving it protected from dust for a minimum of 2 hours and if possible overnight. If speed is essential, it may be dried by warming very cautiously. When the film is thoroughly dry it must be dehaemoglobinized by immersing the slide gently in an upright position in a beaker of clean and preferably distilled water and allowing the haemoglobin to dissolve out. Gentle movement helps, but if the slide is handled roughly the film may become detached. In about 5 minutes, when the film is colourless and opaque, it is removed from the water and allowed to dry. Thereafter it can be stained with Jenner's or Leishman's stain (preferably buffered to pH 7.2), in the same manner as a thin film.

Another method uses a dilute watery solution of Giemsa's stain, which dehaemoglobinizes and stains the film in one operation. The slide with the film facing downwards is placed across 2 thin glass rods

in a flat glass dish and a freshly prepared dilute watery solution of Giemsa's stain (1 drop of stain to 1 ml of water) is run under the slide and left for 15–20 minutes. The slide is then washed gently in a dish of water and allowed to dry in a semi-vertical position. Thick films should be inspected rapidly under the 16 mm objective and then examined systematically under the oil immersion.

Recognition of parasites

The important parasites of the blood are the parasites of malaria, microfilariae of several varieties, trypanosomes, Leishman-Donovan bodies and the spirochaetes of relapsing fever.

Malaria. For the diagnosis of malaria, thick films should be used for the detection of parasites and thin films for their identification. Films should preferably be taken when the patient's temperature is raised. Thick films stained as described should be examined systematically for 10–15 minutes before concluding that no parasites are present. The recognition of parasites in thick films requires practice. White cells, platelets, bacteria, the remains of reticulocytes and miscellaneous dirt can be mistaken for parasites. Parasites have definite morphological and staining properties and objects which do not show these are not parasites.

For details of the identification of the different types of parasites in thin films, larger works must be consulted. The main distinguishing points are as follows (Plate XV).

In infection with *Plasmodium falciparum*, which produces malignant tertian malaria, schizogony generally takes place in the tissues, so that except in rare cases in moribund patients, ring forms and a few crescent-shaped gametocytes only are seen in the peripheral red blood cells. Ring forms of any species consist of a rim of cytoplasm which stains blue and a small nucleus or chromatin dot which stains red. The rings of *Plasmodium falciparum* are usually, though not invariably, small and delicate, and the red cells are not enlarged. More than one ring may appear in a single red cell and some rings may have two chromatin dots. Marginal forms or *formes appliquées* with the parasite lying along the edge of the cell may be seen. A few crescent-shaped gametocytes, which are easily recognized (Plate XV), may be seen in films from untreated patients, but if absent at this time they may appear some 7–10 days after the beginning of treatment.

In the remaining three species, schizogony takes place in the peripheral blood, so that ring forms, large trophozoites and schizonts will be present together in films.

In infections with *Plasmodium vivax*, which produces benign tertian malaria, the rings are large and stout and often measure one-third of the

diameter of the red cell. The red cells may be enlarged, and if properly stained may show well marked Schüffner's dots (Plate XV). Large irregular trophozoites containing brown pigment, and mature schizonts with 12–24 merozoites, may be seen (Plate XV).

In infections with *Plasmodium malariae*, which produces quartan malaria, the ring forms are also large and stout, but the larger trophozoites are more compact and dense-looking and frequently take a characteristic band form (Plate XV). Mature schizonts contain some 8 merozoites arranged in a rosette form around a mass of golden yellow pigment. Further, Schüffner's dots are not seen and the red cells are not enlarged.

In infection with *Plasmodium ovale*, which is much the rarest of the four species, the parasites have some of the characteristics of *Plasmodium vivax* (e.g. large and prominent Schüffner's dots) and others of *Plasmodium malariae* (e.g. compact large trophozoites and occasional band forms). The most characteristic feature is the distortion in shape of the red cells, which become oval or fimbriated, and the schizonts only contain 8–10 merozoites (Plate XV).

In some cases, mixed infections may be present.

Trypanosomiasis. In the diagnosis of trypanosomiasis, examination of the blood is generally less efficient than the examination of fluid obtained by gland puncture. An enlarged gland, usually in the posterior triangle of the neck, is held firmly between the thumb and fingers of the left hand, while a moderate sized hypodermic needle is plunged through the skin and into the substance of the gland. A small amount of gland fluid passes into the needle and suction is neither necessary nor desirable. The needle is withdrawn and its contents blown out on to clean glass slides. The fluid should be examined fresh and unstained as described for fresh blood films and thin films should be stained with Leishman's stain.

Trypanosomes (Plate XV) may also be found in thick or thin blood films and, in advanced cases, in films made from the deposit of centrifuged cerebrospinal fluid. (For methods of concentrating trypanosomes in the blood, larger works must be consulted.)

The important trypanosomes of man are *Trypanosoma gambiense* and *T. rhodesiense*, which cause African sleeping sickness, and, as seen in human blood, are usually morphologically indistinguishable; and *Trypanosoma cruzi*, which causes Brazilian trypanosomiasis (Chagas's disease). The latter exists chiefly in a non-flagellated form in the organs and muscles and only occasionally appears in the blood as a flagellate trypanosome (Plate XV).

In a suspected case of trypanosomiasis, several specimens of gland fluid and of blood should be examined, both fresh and unstained, and

in stained films. Fresh films should be examined under the 16 mm and then under the 4 mm objective. Under the latter trypanosomes may be seen 'lashing' their way amongst the cells and are often first detected by the commotion they produce in the latter. This movement must not be confused with that produced by the organisms of relapsing fever (*Borrelia recurrentis*) (Plate XV).

In stained films, examined under the oil-immersion lens, typical trypanosomes are seen as elongated fusiform structures some 14–30 μm long and 1–3 μm broad with a longitudinal undulating membrane and a terminal flagellum projecting from the anterior end (Plate XV). There is a centrally placed nucleus, and at the posterior end a smaller black-staining kinetoplast. The shorter forms are more stumpy and may have little or no free flagellum.

Kala-azar. In the diagnosis of *kala-azar*, Leishman–Donovan bodies may be looked for in the blood or in material obtained by sternal, gland, spleen or liver puncture. Of these, examination of marrow obtained by sternal puncture (p. 150) is probably the simplest and safest method, but in occasional cases the parasites may be found by the examination of stained blood films, when they are seen in the cytoplasm of large mononuclear cells. When direct microscopic examination fails, culture methods (for which larger works must be consulted) are frequently successful.

The bodies may be seen in thick or thin stained blood films which should be searched systematically under the oil-immersion objective. The parasites are seen as round or oval bodies from 2 to 5 μm in diameter, containing a large round or oval solid-looking nucleus and a smaller more deeply stained and usually rod-shaped kinetoplast. In Leishman-stained films the cytoplasm is blue and the nucleus and kinetoplast are red (Plate XV). Giemsa staining (p. 156) is preferable.

While the term Leishman–Donovan bodies strictly applies to the Indian form of *kala-azar*, exactly similar *Leishmania* may be found in the blood or tissues in cases of Mediterranean *kala-azar*, from fluid obtained by puncture at the margin of the lesion in the various forms of cutaneous leishmaniasis or 'tropical sore', and from the mucous membranes of the mouth, nose or throat in *espundia* or South American leishmaniasis.

Microfilariae. Adult filarial worms or macrofilariae are parasites of the lymphatic system or connective tissues. Their presence is diagnosed by the finding of their larvae or microfilariae in the blood stream. Three main varieties of microfilariae are found in the blood of man. These are:

1. *Filaria bancrofti* (*Wuchereria bancrofti*) which is found in the blood stream in any numbers only at night and which causes filariasis,

characterized by irregular fever, lymphangitis and various forms of elephantiasis.

2. *Filaria loa-loa*, which is found in the blood stream only by day, and which causes loaisis, characterized by transient red painful swellings known as calabar swellings.

3. *Filaria perstans*, which is non-periodic, appearing equally by day and night, and has no recognized pathogenic effects.

If filariasis is suspected, blood should be examined say at 8 a.m., noon and 4 p.m., and again at 8 p.m., midnight and 4 a.m. Fresh unstained films should be used for the detection of the filariae, and stained ones, thick if the larvae are scanty and thin if they are plentiful, for their identification.

In fresh unstained films microfilariae are easily seen under the 16 mm objective, as actively moving linear objects. In stained films they are seen as wormlike objects with a round head and a pointed tail, from 5 to 8 μm broad (i.e. about the diameter of a red cell) and from 100 to 300 μm long (Plate XV). The main differentiating features of the three species, apart from their periodicity, are as follows. *Filaria bancrofti* and *F. loa-loa* have a delicate sheath, which can be seen where it projects beyond the rounded head and pointed tail of the larva, whereas *F. perstans* is unsheathed. All larvae have a central column of nuclei extending from the head more or less to the tail. In *F. loa-loa* and *F. perstans* the nuclei extend to the extreme tip of the tail, whereas in *F. bancrofti* the column ends short of the tip.

Microfilariae do not stain well by Leishman's stain. Thick films should be dehaemoglobinized and stained by Giemsa as described under malaria (p. 156). Microfilariae are readily recognized by the intense staining of their nuclei, and the sheath, if present, is easily seen.

Relapsing fever. The *Borrelia recurrentis* of relapsing fever should be sought under the 4 mm objective in fresh unstained films of blood and in thin films stained by Leishman's method under the oil-immersion. Fresh unstained films should be examined with the light well cut down. Agitation of the red cells usually calls attention to the presence of parasites, which may otherwise be difficult to detect.

In thin stained films the spirochaete is seen as a linear object with tapering ends, 0.4 μm in breadth and 10–30 μm in length. The spiral shape which it possesses in life is lost and the body lies in irregular curves. In searching for the organism, it is important to direct the eyes deliberately to the spaces between the red cells rather than to the red cells, or the parasite may be missed.

THE RESPIRATORY SYSTEM

Anatomical Landmarks — Inspection — Palpation — Percussion — Auscultation — The Sputum — X-Ray Examination — Bronchoscopy, Thoracoscopy and Mediastinoscopy — Pleural Aspiration and Biopsy — Lung Function Tests — Skin Tests

ANATOMICAL LANDMARKS

Lobes of the lungs. It is important to know the limits of the individual lobes of the lungs. A line from the second thoracic spine to the sixth rib in the mammary line corresponds to the upper border of the lower lobe (the major interlobar fissure). A horizontal line on the right side from the sternum at the level of the fourth costal cartilage, drawn to meet the first, marks the boundary between the upper and middle lobes (the minor interlobar fissure). The greater part of each lung, as seen from behind, is composed of the lower lobe, only the apex belonging to the upper lobe; while the middle and upper lobes on the right side, and the upper lobe on the left, occupy most of the area in front. In the axillary regions, parts of all the lobes are accessible.

The bifurcation of the trachea corresponds in front with the lower border of the manubrium sterni, that is with the angle of Louis; behind, with the disc between the fourth and fifth thoracic vertebrae.

The twelfth rib cannot always be felt and so it is not wise to count the ribs from below upwards. These are best counted downwards from the second costal cartilage. This cartilage articulates with the sternum at the extremities of the angle of Louis, a transverse bony ridge at the junction of the body and the manubrium, which is easily felt beneath the skin.

Anatomy of the bronchi. The two main bronchi each give off four main branches: on the right, one to the upper lobe, one to the middle lobe, one to the dorsal lobe (the upper and posterior part of the lower lobe), and one to the remainder of the lower lobe; and on the left, one to the upper lobe proper, one to the lingular process of the upper lobe (which represents the middle lobe on the left side), and a dorsal and lower lobe bronchus as on the right side. These main bronchi then divide into segmental bronchi, which supply individual segments of lung. It is important to have a working knowledge of these segments, because it is often possible from the signs and X-ray appearance present to deter-

mine which segment and which segmental bronchus is affected by disease. The accompanying diagrams (Figs 36–38), give a simplified scheme of the anatomy of the segmental bronchi and indicate the ‘respiratory districts’ or bronchopulmonary segments supplied by them.

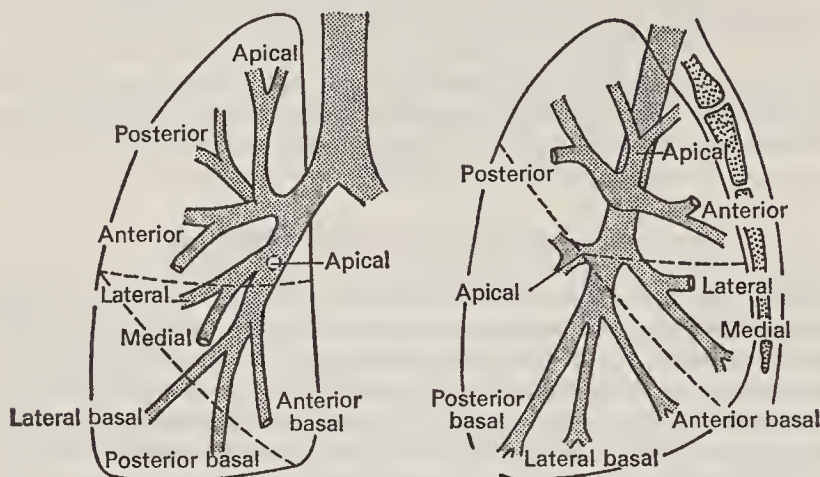


Fig. 36. The right lung (anterior and lateral aspects) showing the segmental bronchi.

- | | | |
|-------------|---|--|
| Upper lobe | { | Apical bronchus and segment |
| | { | Posterior bronchus and segment |
| | { | Anterior bronchus and segment |
| Middle lobe | { | Lateral bronchus and segment |
| | { | Medial bronchus and segment |
| | { | Apical bronchus and segment |
| | { | Medial basal bronchus and segment (bronchus not shown) |
| Lower lobe | { | Anterior basal bronchus and segment |
| | { | Lateral basal bronchus and segment |
| | { | Posterior basal bronchus and segment |

INSPECTION

Form of the chest

The shape of the chest varies with the build of the individual, often being short, broad and deep in the thick-set and long, flat and narrow in the tall and spare. There is also wide variation in the thickness of the muscles and the slope of the shoulders in healthy people. Estimates of the significance of variations in the shape of the chest must, therefore, take into account the build of the individual. Normally the chest is bilaterally symmetrical and on cross-section is elliptical. Its shape is

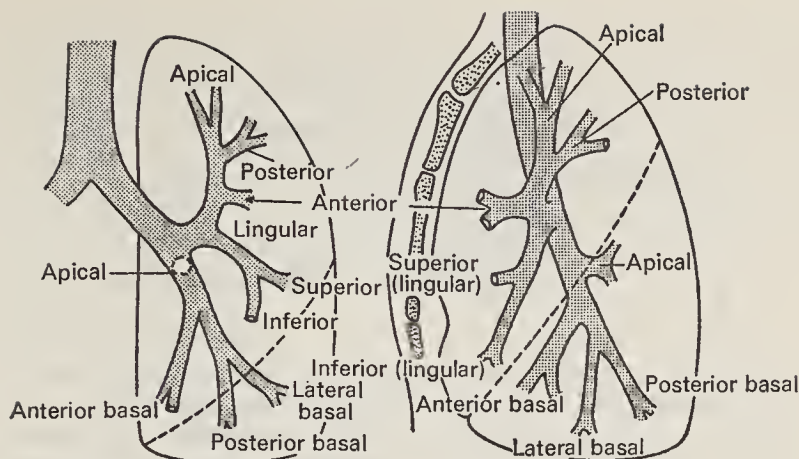


Fig. 37. The left lung (anterior and lateral aspects) showing the segmental bronchi.

Upper lobe	Upper division bronchus	Apical bronchus and segment
		Posterior bronchus and segment
	Lingular (lower division) bronchus	Anterior bronchus and segment
		Superior bronchus and segment
Lower lobe		Inferior bronchus and segment
		Apical bronchus and segment
		Anterior basal bronchus and segment
		Lateral basal bronchus and segment
		Posterior basal bronchus and segment

affected by extrapulmonary as well as pulmonary disease. For example, in the past, severe rickets was a common cause of chest deformity, the combination of softness of the bones and obstruction to respiration (due to adenoids and chronic or recurrent upper respiratory tract infection) leading to the deformities known as pigeon-chest and Harrison's sulcus.

Disease of the vertebral column can produce *kyphosis* (forward bending) or *scoliosis* (lateral bending), which often occur together. These lead to obvious asymmetry of the chest and may decrease the size of the thoracic cage and restrict lung movement. Scoliosis may also lead to clinical and radiological displacement of the trachea and apex beat. The spine and rib cage may become particularly immobile in ankylosing spondylitis, which may lead to a fixed kyphosis.

Pulmonary disease produces several important deformities of the chest. Unilateral apical fibrosis due to tuberculosis may cause obvious flattening of one apex, whilst more extensive unilateral fibrosis or collapse in childhood can even lead to scoliosis. Severe obstructive

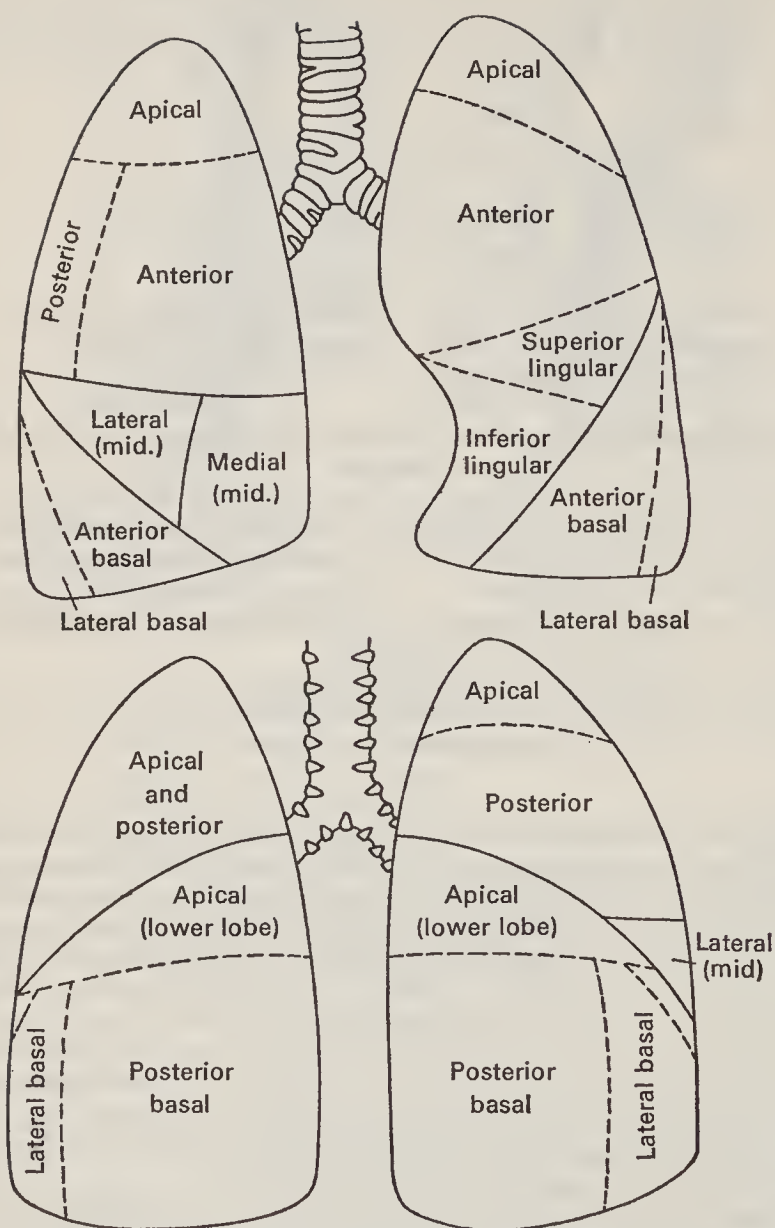


Fig. 38. The respiratory districts supplied by the segmental bronchi.
Above, anterior aspect. Below, posterior aspect.

airways disease (p. 181) leads to a *barrel-shaped chest*. There is obstruction to expiration, and the lungs become overinflated, so that the chest becomes fixed in what is normally the position of full inspiration. Since no further expansion is possible, the rib cage tends to move up in one piece by the action of the accessory muscles of respiration, which become more prominent than usual. The ribs are less obliquely set than usual, the anteroposterior diameter of the chest is increased, the spine becomes unduly concave forwards and the sternum is more arched than usual, with a prominent angle of Louis.

Movements of the chest

The *rate of respiration*, for a normal adult at least, is about 14–18/minute, but up to 22 if the patient is nervous under examination. An increased rate of respiration or *tachypnoea* is an important sign of pulmonary disease, which is frequently missed owing to lack of observation or inaccurate counting. Increased rate may result from exertion, nervous excitement, fever or hypoxia. Such hypoxia may be due to cardiac, pulmonary, bronchial or laryngeal causes, to alteration in the oxygen-carrying power of the blood or to interference with the normal reflex control of respiration. Tachypnoea may also arise from the association of pain with breathing, as in pleurisy and peritonitis, when the breathing becomes shallow and must therefore be more frequent to make up for the slighter expansion.

The *ratio between respiration and pulse* in health is about 1 to 4. In severe pneumonia respiration may occur almost as frequently as the pulse. In certain cases of narcotic poisoning, the ratio may become 1 to 6 or 7.

The rhythm varies very considerably even in health and if the act is performed consciously it may become very irregular. Study it, therefore, when the patient is off his guard, as only then can accurate observations be made. Either inspiration or expiration may be unduly prolonged, the former being commonly associated with laryngeal or tracheal diseases, the latter with bronchial or pulmonary. A peculiar type, where successive respirations gradually get deeper and deeper till a maximum is attained, and then fall off again until a pause of complete apnoea occurs, to be followed by another wave of gradually deepening and then diminishing respiration, is known as *Cheyne–Stokes breathing*. The pause may last for fully half a minute, though it is often shorter, and the whole cycle is usually completed in less than two minutes. It is most conspicuous when the patient who exhibits it is asleep or unconscious, but may be overlooked if the patient is awake, and particularly if he is talking. Cheyne–Stokes breathing occurs most commonly in cardiac and renal failure, severe pneumonia, increased intracranial pressure and narcotic drug poisoning.

Movement. Note its amount, whether it is expansile in character, and whether it is similar, or different, on the two sides and over corresponding areas. Movement and expansion are not interchangeable terms; in emphysema the chest may move considerably, but there is little expansion. Chest expansion in men can be measured with a tape measure round the chest just below the nipples. In a fit young man it may be 5–8 cm; in emphysema it may be 1 cm or less.

Diminished or absent expansion may be due to pleurisy with effusion, pneumothorax, consolidation, collapse, fibrosis or the presence of a neoplasm. It is also found in tuberculosis and lobar pneumonia, the former especially at the apices, the latter at the apex or base according to the situation of the disease.

PALPATION

Before making a systematic examination, it is well to lay the hand on any part of the chest which presents an obvious swelling, or where the patient complains of pain. Look at the face rather than the part under examination, so as to avoid causing unnecessary pain. Pain may be due to recent injury to the chest wall, or to inflammatory conditions; to intercostal muscular pain, where, as a rule, specially painful spots can be discovered on pressure; to a painful costochondral junction; to secondary malignant deposits in the ribs; to herpes zoster, before the appearance of the eruption; or to pleurisy. In the case of pleurisy, pressure may considerably increase the pain. Pain may also be due to cardiac causes, such as coronary thrombosis and pericarditis. At the same time the nature of any swelling should be investigated. Fluctuation occurs when an abscess has formed in the chest wall.

The positions of cardiac impulse and trachea should then be determined. Feel for the trachea in the suprasternal notch and decide whether it is placed centrally or deviated to one or other side, by its relation to the suprasternal notch and the insertion of the sternomastoids. Slight deviation of the trachea to the right may be found in healthy people. Displacement of the cardiac impulse alone may be due to scoliosis, the commoner form with its convexity to the right causing a displacement of the cardiac impulse to the left and vice versa; to funnel depression of the sternum or to enlargement of the left ventricle, particularly in aortic regurgitation and hypertension. In the absence of these conditions, a significant displacement of the cardiac impulse or trachea, or of both together, suggests that the position of the mediastinum has been altered by disease of the lungs or pleura. The main conditions which 'push' the mediastinum away from the affected side are pleural effusion and pneumothorax, and the main conditions which draw it towards the affected side are fibrosis of the lung in tuberculosis or after broncho-

pneumonia, and collapse of one or more lobes. In fibrosis or collapse of the upper lobe of a lung, the trachea only is displaced.

The *nature of the respiratory movements* must next be studied. It is important to make certain that the two sides of the chest move to approximately the same extent. This is done by fixing the fingertips of either hand at the patient's sides and making the tips of the thumbs just meet in the middle line in front of the chest. The patient is directed to take a full inspiration, when the distance of departure of the thumbs from the middle line indicates the extent of expansion of either half of the chest. The causes of diminished expansion have been mentioned under inspection.

Vibrations may be detected by palpation. For this purpose the palm of the hand should be applied flat on the chest. The patient is then told to repeat 'one, one, one', or 'ninety-nine', in a clear voice. The hand placed on the thorax detects distinct vibration whilst this is done and one must determine whether the vibrations in corresponding areas on the two sides of the chest are approximately equal in intensity—not, however, forgetting that where the heart encroaches on the left lung the fremitus is necessarily much diminished. *Vocal fremitus is increased* when the lung is consolidated, or contains a large cavity near the surface. *Vocal fremitus is diminished* when the corresponding bronchi are obstructed or totally absent when the lung is separated from the chest wall by a pleural effusion. The cause in this case is not that fluid is a bad conductor of sound or of vibration—the reverse is the case—but that the collapsed lung itself fails to convey the vocal fremitus and so the vibrations never reach the fluid. In young persons and women, the vocal resonance is different both in character and intensity from that in male adults.

PERCUSSION

Method of percussion. The middle finger of the left hand is placed firmly on the part which is to be percussed. The back of its middle phalanx is then struck with the tip of the middle finger of the right hand. The stroke should be delivered from the wrist and finger joints, not from the elbow, and the percussing finger should be so bent that when the blow is delivered its terminal phalanx is at right angles to the metacarpal bones and strikes the pleximeter fingers perpendicularly. As soon as the blow has been given, the striking finger must be raised, just as the hammers of a piano fall back from the wires as soon as these have been struck. The blow should be no heavier than is necessary to elicit the resonance of the part being examined, and the wrist joint must move loosely. Repeated heavy blows cause much discomfort to sensitive patients. Sir Robert Hutchison in his rich Scottish accent was heard to say to a student: 'Young man, I see you are one of those who will go through life leaving a tr-r-rail of br-r-roken r-r-ribs behind you.'

The *character of the sound produced* varies quantitatively and quali-

tatively. When the air in a cavity of sufficient size and appropriate shape is set into vibrations which are not modified by excessive tension of the containing walls of the space, the sound heard has a tympanitic character; such a note is heard on percussion over an air-containing viscus, such as the stomach; but when the cavity is subdivided into a number of small loculi by numerous septa, more or less tense, a characteristic resonance, no longer tympanitic, is produced. Such conditions prevail in the healthy lung and the observer must learn by practice to recognize its distinctive quality. In general terms, this pulmonary resonance is low in pitch and clear in character.

Beginning in front, the examiner should tap lightly and directly (i.e. without pleximeter finger) on the most prominent point of each clavicle—being sure that the points examined correspond exactly with each other—and should observe the quality of the sound, and particularly whether the effects on the two sides are identical. Thereafter the other corresponding areas on either side should be carefully percussed in the manner already described. The presence of the heart will interfere, in parts of the left side, with the development of a sound resembling that from the corresponding point on the right. The back and axillary regions should then be examined in the same manner.

It is essential in all parts of the examination that the patient's attitude is a comfortable one and that his arms and shoulders are placed symmetrically, whether he is sitting up or lying down. When in the case of a very ill patient it is only possible to examine the back by rolling him on to his side, only gross differences in the note on the two sides are significant. If possible the patient should be examined lying first on one side and then on the other. Should the patient's chest be asymmetrical, from scoliosis or other cause, equal resonance on the two sides is not to be expected and again only gross differences between the two sides are significant.

The observer should have two objects in mind; first to make a comparison of the percussion note in comparable areas on the two sides; and second, to map out the limits of lung resonance particularly at the apices, the bases, and the area of cardiac dullness.

The *normal degree of resonance* varies from individual to individual and in different parts of the chest in the same individual, being most resonant below the clavicles and scapulae where the muscles are relatively thin, and least resonant over the scapulae.

The lower limits of lung resonance should be determined by percussing from above downwards. The *lower border of the right lung* lies over the liver and is thin; therefore its exact situation is best made out by light percussion. Posteriorly, however, the muffling due to the thick muscles and fat of the back makes it necessary to percuss more firmly. When the patient is obese, very heavy percussion with several fingers

may be necessary. In quiet respiration and on light percussion the lower border is found to lie in the mammary line at the sixth rib, in the mid-axillary line at the eighth rib, in the scapular line at the tenth rib. On heavier percussion some loss of resonance, due to the underlying liver and diaphragm, is found at higher levels and in the mammary line can be detected from the fourth interspace downwards.

On the left side the lower border overlaps the stomach and so the transition is not from lung resonance to dullness, but to tympanitic stomach resonance. Posteriorly, however, the splenic dullness and the dullness of the various solid structures which lie below the lung near the spine are interposed, so that the conditions resemble those found on the right. The position of the lower border corresponds pretty closely with that on the right side; it may, however, be found a trifle farther down.

In disease the resonance may be increased or decreased.

Resonance is increased when the pleural cavity contains air and the lung is more or less collapsed towards the hilum. The note varies from one that is hyperresonant to one that is distinctly tympanitic, according to the amount of air in the pleural cavity.

A characteristic form of high-pitched tympanitic resonance, the *bruit d'airain* (*airain* = brass), 'bell-sound' or 'coin-sound', may also sometimes be heard in pneumothorax, by percussion over the front of the chest with a couple of coins—one being used as a plexor and the other as a pleximeter—whilst the observer listens with the stethoscope at the back of the patient. Failure to elicit the *bruit d'airain* does not mean, however, that a pneumothorax is not present.

Though a definite tympanitic or hyperresonant sound is regularly found in pneumothorax, it is not consistently found in any other pulmonary conditions. It may be found over some large cavities. It is often stated that resonance is increased in emphysema. When, however, a group of doctors at the Royal Postgraduate Medical School were asked to percuss the chests of patients with emphysema and those without chest disease, without knowing the diagnosis, several normal chests were described as being hyperresonant. In the diagnosis of emphysema it is more important to note the increased limits of resonance with loss of liver and cardiac dullness than the quality of the resonance.

Resonance is diminished when the pleura is thickened, when the underlying lung is more solid than usual for any reason and when the pleural cavity contains fluid. Thus, there may be slight impairment of resonance at one apex in a case of pulmonary tuberculosis due to local infiltration and fibrosis. Considerable impairment may be found over areas of lung affected by fibrosis or collapse. Percussion over a completely consolidated lobe produces a definitely dull note, whilst absolute dullness along with a peculiar sensation of resistance in the percussing

finger, so-called 'stony dullness', is the characteristic finding over a pleural effusion of any size. In heart failure, impaired resonance or dullness may be found at the bases of both lungs, indicating oedema of the bases or bilateral effusions.

Myotatic irritability. In certain conditions the muscles on the front of the thorax are unduly irritable, and a light tap over the sternum produces contractions, at some distance off, in the pectoral muscles. This phenomenon occurs in any wasting disease, and is known as myotatic irritability or myoidema.

AUSCULTATION

Three observations must be made at each point auscultated: first, the character of the breath sounds; second, the character of the vocal resonance; and third, the presence or absence of other sounds.

For good auscultation, a patient in bed should lean back against pillows, or lie on his back, and be completely relaxed. To examine the back, the patient should sit up, but if he is unable to do this he should be rolled round first to one side then to the other. In serious cases, the minimum necessary examination should be done. Take care that the chest-piece is accurately applied, that it is not allowed to move on the surface of the skin, and that no undue pressure is exerted. The patient should breathe with his mouth open, regularly and fairly deeply, but not noisily. It is quite useless to attempt auscultation of a patient who is shivering.

Character of respiratory sounds

There are two typical varieties of breath sounds, both of which are audible in health at certain parts of the chest. The first is known as vesicular breathing, the second as bronchial. Vesicular breath sounds are produced by the passage of air in and out of normal lung tissue, and are heard all over the chest under normal conditions. Bronchial breath sounds are produced by the passage of air through the trachea and large bronchi. Under normal conditions they can be heard by listening over the trachea, but they are not heard over normal lung tissue (except where they may modify the sounds heard over normal tissue situated near the trachea and large bronchi, as will be mentioned later). In disease, however, these sounds may be conducted from the bronchi to the chest wall, as, for instance, when a whole lobe is consolidated by pneumonia. Under these circumstances no air enters or leaves the alveoli and no vesicular breath sounds can be heard. Provided, however, that the bronchi are patent, so that bronchial sounds are produced and conducted through them, and provided that sufficient lung is consolidated to convey these sounds to the chest wall, bronchial breathing will be heard over the area affected by these changes.

In *vesicular breathing*, which can be heard typically in the axillary and infrascapular regions of a healthy individual, the inspiratory sound is fairly intense, and is audible during the whole of the act. The pitch is low and the quality is characteristic, being somewhat rustling. The expiratory sound follows that of inspiration without a distinct pause. It only remains audible during the earlier part of the expiratory phase, and under normal conditions the inspiratory sound is heard for at least twice as long as the expiratory.

To learn to recognize *bronchial breathing* the student should listen over the trachea, though he must not expect to hear so intense a type of bronchial respiration when he subsequently examines a diseased lung. The inspiratory sound is moderately intense. It becomes inaudible shortly before the end of inspiration. Its quality is harsh and aspirate. The expiratory sound is generally more intense than the inspiratory; the pitch is often higher; the duration extends through the greater part of expiration, being as long as, or even longer than, the inspiratory sound. In quality it exactly resembles the inspiratory sound, being aspirate in character. Bronchial breathing can most readily be recognized by the quality of the expiratory sound and the definite gap between inspiration and expiration. Some prolongation of the expiratory sound is characteristic of asthma and emphysema, and this must not be mistaken for bronchial breathing. It is due to the fact that in these diseases the act of expiration is itself performed more slowly than in health.

When breath sounds in a superficial bronchus can be heard through normal lung, the sound of the breathing combines both vesicular and bronchial elements. This variety of breath sound is known as *bronchovesicular*, and it is usually the expiratory sound which has more of a bronchial character. It may occur in health near the roots of the lungs behind; in the upper portions near the middle line in front; and especially at the right apex for a few centimetres below the clavicle in front and above the level of the spine of the scapula near the midline behind. These findings, which sometimes lead to a mistaken diagnosis of disease at the right apex, are due to the fact that the trachea lies in immediate contact with the apex of the lung on the right side, whereas it is separated from it on the left by the aorta, the internal carotid artery and the oesophagus.

The breath sounds must be auscultated in the various regions that have already been examined by percussion, their character in each noted and similar regions on the two sides of the chest compared, care being taken that the points examined correspond accurately to one another.

If the student understands how vesicular and bronchial breath sounds are produced, he should have no difficulty in explaining the typical findings in disease. Vesicular breath sounds may be present but reduced in intensity in any condition in which the entry of air to that part of the lung is diminished, as, for instance, in bronchopneumonia,

where some alveoli are affected and others not. Breath sounds of any kind may be diminished or absent where thickened pleura, pleural effusion or pneumothorax interferes with or prevents the conduction of these sounds to the chest wall. They may also be absent in any condition, such as collapse or fibrosis, in which no air enters or leaves alveoli, but at the same time the conditions necessary for the conduction of bronchial breath sounds to the chest wall are not fulfilled. Finally, bronchial breath-sounds may be heard whenever patent bronchi are connected to the chest wall by a sufficiently uniform sound-conducting medium. This occurs classically over consolidated lung, occasionally in the presence of a large cavity, and very rarely over a pleural effusion.

Vocal resonance

The second series of observations is directed to *the intensity and character of the vocal resonance*. It varies in intensity even in health being more intense the nearer the stethoscope is to the larger bronchi. When the patient repeats the words 'one, one, one', or 'ninety-nine', the ear perceives, not the distinct syllables, but a resonant sound, the intensity of which depends on the loudness and depth of the patient's voice and on the conductivity of his lungs.

Each point examined on one side of the chest should be at once compared with the corresponding point on the other side. Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest-piece of the stethoscope. If it seems to be nearer the ear than this, the resonance is increased. When it appears to be near the earpiece of the stethoscope the increase is marked and the condition is often described as *bronchophony*.

If the words become clear and seem to be spoken right into the auscultator's ear, it will generally be found that whispered words are distinctly heard. This condition is called *whispering pectoriloquy*. Increased resonance occurs when the lung substance conducts the sound waves set up by the voice more clearly than usual from the bronchi. Consolidation is the commonest cause. Bronchophony and whispering pectoriloquy occur when a moderately large bronchus is surrounded by layer of solid lung reaching to the chest wall, as in lobar pneumonia. Whispering pectoriloquy is also fairly characteristic of a cavity of some size communicating with a bronchus and may be heard above the level of a pleural effusion. In some cases a certain degree of pectoriloquy is heard in health in the proximity of the trachea and large bronchi and particularly at the right apex.

For reasons already explained, vocal resonance is either entirely abolished or much diminished where a layer of fluid separates the lung from the chest wall (except when bronchial breathing is heard, see above) and in pneumothorax. It is also diminished in cases of thickened pleura and of emphysema.

Above the level of a pleural effusion or in some cases over an area of consolidation, a nasal or bleating character may be imparted to the voice. It is known as *aegophony*.

Added sounds

These may arise in the lung or in the pleura. Sounds resembling pleural friction may be produced by movement of the stethoscope on the patient's skin, or of the observer's hands or clothes against the stethoscope. Sounds arising in the patient's muscles may resemble adventitious sounds and in particular the shivering of a cold patient makes any attempt at auscultation useless. The application of the stethoscope to hairy skins may produce sounds indistinguishable from crepitations. Sounds resembling coarse crepitations may also be heard over a broken rib.

The nomenclature of the added sounds arising in the lung has been confused since their original description by Laennec. He introduced the French word *râle* to describe any added sound heard in the chest. When he wrote or spoke in front of patients, he used the Latin word *rhonchus* in the same connotation. As a result of confusion in translation, the terms are often used differently today. The least confusing classification of added sounds is to divide them into continuous wheezing sounds, or *rhonchi*, and interrupted bubbling or crackling sounds, or *crepitations*.

Rhonchi, which are prolonged uninterrupted noises, arise in the bronchi and are due to partial obstruction of their lumen, by swelling of the mucosa, by viscid secretion or by constriction of bronchial smooth muscle. They may be high or low pitched, depending on whether they arise in small or large bronchial tubes. High-pitched rhonchi are called *sibilant* and have a squeaky quality; low-pitched rhonchi are called *sonorous* and have a snoring quality. They may also be palpable. Rhonchi are characteristic of bronchitis and asthma, and in the latter condition are high-pitched and expiratory. When they are localized to one side of the chest, the possibility of a localized obstruction of a bronchial tube should be considered, as for example in bronchial carcinoma. The word 'bronchospasm' as a description of a physical sign should be avoided.

Crepitations are discontinuous crackling or bubbling sounds which may be produced in the alveoli, the bronchi or the cavities. They sound like the bursting of air bubbles, and indicate the presence of fluid secretions. They may be classified as fine or coarse.

Fine crepitations are probably caused by the opening up of collapsed alveoli. The separation of the walls is accompanied by a clicking sound and when this condition occurs in a number of alveoli, the combined effect is to produce a sound of fine crepitation, which can be imitated by rolling a few hairs between the finger and thumb in front of the ear. It occurs only near the end of inspiration and indicates the presence of exudate in the alveoli of the affected part of the lung. Fine crepitations

are characteristically present during the first stage of pneumonia, at the apices in tuberculosis and at the bases in heart failure.

Coarse crepitations are bubbling or clicking noises which can probably arise in many sites, including large and small branches of the bronchial tree or in cavities, and may be heard at almost any phase of, or during the whole of, respiration. They are often present in bronchitis in association with rhonchi: however, when they are restricted to the lung bases, the possibility of bronchiectasis or fibrosing alveolitis should be considered.

A few crepitations may be heard in health, particularly at the lower borders of the lungs. These are abolished if the patient is asked to cough and are of no significance. In other cases crepitations are intensified after a cough or may only then make their appearance. Crepitations brought out by coughing are known as *post-tussive crepitations*. They are an important sign of tuberculous infiltration, and may also be heard over cavities.

A *friction sound* or *pleural rub* is characteristic of pleurisy at the stage when exudation is not abundant enough to separate the inflamed and roughened pleural surfaces. It has a creaking or rubbing character, often quite characteristic, but sometimes rather hard to distinguish from a crepitation. The friction sound may be fine or coarse. In some instances it is palpable, but since coarse crepitations may be so too, this does not distinguish them. The chief features of difference are that friction sounds occur during that part of inspiration when the roughened surfaces are rubbing against each other, to reappear at a corresponding period of expiration. They are, moreover, unchanged after the patient has coughed, whereas crepitations may alter under these conditions, because of changes in disposition of the secretion which causes them.

Finally there are certain manifestations of pulmonary disease which should be looked for outside the chest itself. These include clubbing of the fingers and cyanosis (both mentioned in previous chapters), and enlargement of cervical or axillary lymph nodes or the liver from secondary carcinoma. Respiratory failure with carbon dioxide retention (p. 182) may be associated with certain neurological signs. These including a coarse flapping tremor of the hands (asterixis), generalized twitching movements and even convulsions. Such patients may become drowsy or stuporose and this is aggravated by sedatives or injudicious oxygen therapy. In some cases there is papilloedema, which in a few instances may even be followed by optic atrophy.

THE SPUTUM

Sputum may be mucoid, purulent or frothy. Any of these varieties may contain blood, or the sputum may consist entirely of blood.

Mucoid sputum occurs characteristically in chronic bronchitis when secondary infection is not present. It is clear, tough and sticky and usually scanty. Particularly tenacious sputum may be found in asthma. This may block the bronchi by forming plugs, or else be coughed up as 'casts' of the bronchial tree—during or after an attack; alternatively, sticky particles like sago—the *perles* of Laennec—may be expectorated after an attack.

Mucopurulent sputum is seen in bronchitis (or other upper respiratory infections) when secondary bacterial infection has occurred: the sputum in bronchitis may also be frankly purulent.

Purulent sputum is thick and yellow (or green) and not sticky. It may occur in any condition in which infection is present and is characteristic of bronchiectasis, bronchopneumonia and lung abscess. In bronchiectasis and lung abscess it may be copious and its expectoration may be readily influenced by change of posture.

Frothy sputum, characteristic of pulmonary oedema, may be white or pink and is often copious.

Blood may be coughed up alone, or the sputum may be more or less blood-stained. It must be distinguished from blood brought into the mouth from epistaxis or haematemesis. Its brighter colour and its frothy appearance usually makes its origin obvious. Further, patients who have had a haemoptysis commonly bring up blood-stained sputum for a day or two, while bleeding from the upper intestinal tract is characteristically followed by melaena. Haemoptysis may be due to pulmonary causes, including tuberculosis, bronchiectasis, pulmonary embolus and carcinoma; to cardiac causes, including mitral stenosis; and very rarely to aneurysm of the aorta.

Several diseases cause a characteristic colouration of the sputum. In lobar pneumonia it may be rusty and so viscid that it often will not fall out of an inverted spittoon; it is bright yellow or green when a liver abscess has been ruptured into the lung, and the latter colour also appears in some cases of pneumonia. When an amoebic hepatic abscess has discharged into the lung the sputum has the appearance of anchovy sauce.

The quantity of sputum coughed up in twenty-four hours is important; and especially whether change of position produces a large quantity. Bronchitic patients bring up most sputum during the first two hours after waking in the mornings.

The odour of the sputum is rarely important, but it may be putrid in bronchiectasis or lung abscess.

Microscopical examination of sputum

The principal value of microscopical examination of the sputum is in the detection of bacteria and in the recognition of malignant cells.

Eosinophils may be found in the sputum in allergic conditions, e.g. some cases of asthma, and in pneumonia due to parasitic worms and in aspergillosis.

Notes on bacteriological examination of the sputum will be found on p. 335.

Malignant cells may be seen in the sputum, particularly in patients with squamous carcinomas of the main bronchi. Skill is required to differentiate them from epithelium and other cellular debris, but their appearance is sometimes unmistakable (Plate XII).

Less common constituents of the sputum include fungi and yeasts, and golden-yellow asbestos bodies in asbestosis (Plate XII).

X-RAY EXAMINATION

Radiological examination of the chest is most important, because many localized and even some widespread infiltrative lesions (e.g. sarcoidosis) may produce no abnormal physical signs. It is paramount, in the early diagnosis of tuberculosis and carcinoma. Serial X-rays form an integral part of the estimation of progress in many chest diseases. A brief outline of the standard methods will be given here.

Radiography

The ordinary standard X-ray film of the chest is a *postero-anterior* view, that is to say one taken with the film against the front of the patient's chest and the X-ray tube 2 metres behind the patient. It is examined systematically on a viewing box in every case. The following is a simple plan of examination.

1. The bony skeleton. Is the chest symmetrical? Is any scoliosis present? Are the ribs unduly crowded or widely spaced in any area? Are cervical ribs present? Are the ribs eroded or do they appear the site of malignant deposits?

2. The position of the patient. Is the patient straight or rotated? If straight, the inner ends of the clavicles will be disposed symmetrically with reference to the vertebral column.

3. The position of the trachea. This is seen as a dark column representing the air within the trachea. The cartilagenous rings are not visible. Is it centrally placed or deviated to one or other side?

4. The outline of the heart and mediastinum. Is this normal in size, shape and position?

5. The diaphragm. Can the outline of the diaphragm be seen on each side, and is it normal in shape and position? Are the cardiophrenic and costophrenic angles clearly seen?

6. The lung fields. For radiological purposes these are divided into three zones:

Zone 1 (upper zone) extends from the apex to a line drawn through the lower borders of the anterior ends of the second costal cartilages.

Zone 2 (mid zone) extends from this line to one drawn through the lower borders of the fourth costal cartilages and contains the hila of the lungs.

Zone 3 (lower zone) extends from this line to the bases of the lungs.

Each zone is systematically examined on the two sides and any area which appears abnormal is carefully compared with the corresponding area on the opposite side. The minor interlobar fissure, which separates the right upper and middle lobes, may sometimes be seen running horizontally in the third and fourth interspace on the right side. The major interlobar fissure, which separates the lower lobes from the remainder of the lungs, is not seen in a normal postero-anterior film.

Lateral views are indispensable in the localization of lung lesions for the postero-anterior view does not show whether a shadow is situated in the anterior or posterior part of the chest, or (if in the mid zone) whether in the upper or lower lobe.

The following is a simple plan of examinations.

1. The bony skeleton.

2. The position of the trachea.

3. The diaphragm. As the level differs on the two sides, a double outline may be seen, that of the side nearer the film being the clearer.

4. The lung fields. These are obscured by two relatively opaque areas, one above and behind, due to the shoulder joint, and one below and in front, due to the heart, which rests on the anterior part of the diaphragm. There are thus left two relatively clear areas—one above and in front, behind the upper part of the sternum, and one below and behind, including the angle between the diaphragm and the spine.

In the lateral views, the interlobar fissures are more often seen. Their normal positions have already been described (p 161). Their recognition is useful both in localizing lesions and in detecting shrinkage of a lobe from fibrosis or collapse.

Screening

This procedure is used mainly to detect abnormalities of the heart and paralysis of the diaphragm. By standing the patient in different posi-

tions, it is possible to see enlargement of the various chambers of the heart and main vessels and paradoxical pulsation of the left atrium. Enlargement of the left atrium is detected by noting displacement of the barium-filled oesophagus. When a diaphragmatic paralysis is present *paradoxical* movement is seen when the patient coughs or sniffs, the diaphragm ascending when it should descend, and vice versa.

Bronchography

For this purpose a radiopaque iodized oil (usually Dionosil Oily) is introduced into the trachea and allowed to run into the bronchi. X-ray pictures are taken and the corresponding bronchi are clearly outlined. With suitable manipulations, the whole of the bronchial tree can be outlined, but more than one sitting may be required.

The oil may be introduced:

1. By passing a special needle through the cricothyroid membrane, after suitable anaesthetization of the needle track and the mucous membrane of the trachea (this method is the one most usually adopted nowadays).

2. By means of a catheter passed through the nasal cavity into the trachea.

3. Directly into the larynx over the back of the tongue.

4. Through a bronchoscope (this method is used only occasionally and for special purposes).

If the bronchi on both sides are to be outlined at one sitting, the side thought to be abnormal should be filled first, and postero-anterior and lateral views taken. The opposite side can then be filled and a further postero-anterior view taken. The patient should be warned not to attempt to swallow food or drink till the effect of any local anaesthetic given has worn off.

Tomography

An ordinary X-ray picture consists of shadows at all depths in the chest, superimposed on one another. It has the disadvantage that not more than some 40% of the lung tissue is shown without its being obscured by shadows of the bony thorax or of mediastinal contents. The tomograph is a device whereby a picture is obtained of a section of the thorax at any given depth. The tube and plate are moved in the arc of a circle as the exposure is made, in such a manner that the structures in one section only remain in focus and anything out of the plane of this section is blurred out. Sections can be taken at different depths in the chest, as desired, and so the appearances in chest X-rays can often be greatly simplified. It is mainly used to detect cavitation in apparently opaque shadows in the lung fields, and to give a clearer picture of hilar

shadows. It may also indicate narrowing of a bronchus due to lesions such as carcinoma.

BRONCHOSCOPY, THORACOSCOPY AND MEDIASTINOSCOPY

By means of the bronchoscope, the main bronchi and their branches can be directly inspected, small portions of tissue can be removed for biopsy, and therapeutic procedures carried out. The main value of bronchoscopy is in the diagnosis of carcinoma of the bronchus and in deciding whether this is operable. After an artificial pneumothorax has been induced, the pleura can be inspected with the aid of a thoracoscope and further assistance in diagnosis may be obtained. These are specialized surgical techniques. Mediastinoscopy is a recently introduced procedure. An instrument is inserted behind the sternum, which enables the observer to inspect structures such as glands and to take specimens for biopsy.

PLEURAL ASPIRATION AND BIOPSY

Pleural effusions may be drained by inserting a wide-bore needle into the fluid-filled pleural space through one of the rib spaces. This is done under local anaesthesia, and the needle is inserted perpendicular to the skin and pleura, just above a rib margin. Aspirations may be *therapeutic*, to relieve respiratory embarrassment due to a large effusion, or *diagnostic*, to remove fluid for examination.

Pleural fluid can be examined macroscopically and microscopically. Full details cannot be given here, but the colour, consistency and quantity should be noted. A *transudate* from the capillaries, as occurs in cardiac and renal disease, can be distinguished from an *exudate* resulting from pleural inflammation by its lower protein content (< 3.0 g/100 ml) and specific gravity (usually < 1.015). Frankly blood-stained effusions occur with carcinoma, pulmonary infarction or trauma. In tuberculosis effusions the fluid is straw-coloured and copious (often well over 1 litre) and may coagulate on standing; under the microscope many leucocytes are seen, lymphocytes often predominating. Tubercle bacilli are rarely seen, but can more commonly be cultured. In other inflammatory exudates, many polymorphs are seen. In *empyema* pus is aspirated which may be full of white cells and organisms.

Needle biopsy of the pleura may also be useful in establishing the diagnosis of the cause of a pleural effusion. In a high proportion of patients the presence of carcinoma or tuberculosis may be detected. Drill biopsy of lung can also be performed and is useful in establishing

a histological diagnosis in *diffuse* lung diseases, such as fibrosing alveolitis.

LUNG FUNCTION TESTS

In recent years, tests of lung function of increasing complexity have been introduced, which are beyond the scope of this chapter; but some of the simple tests useful in clinical practice will be described. It must be stressed that lung function tests enable the clinician to make a *physiological* rather than a *pathological* diagnosis. That is to say, they will, for instance, tell one that there is obstruction to air flow, but not that the patient has a bronchial carcinoma. Also, they are more likely to be abnormal if there is a diffuse process affecting the lung than if there is merely a localized lesion. They may be useful under the following circumstances:

1. To give an objective assessment of a patient's disability.
2. To follow the progress of a disease and the effect of treatment.
3. To try to differentiate possible causes of a patient's dyspnoea.
4. To aid the management of cases of respiratory failure.

Vital capacity and spirometry

The simplest and still perhaps the most valuable tests are the measurement of the patient's *vital capacity* and the recording of the *expiratory spirogram*. These can be measured by various types of *spirometer* and recorded graphically (Fig. 39). The patient inhales maximally and then breathes out as hard and as fast as he can. Measurements are then made of the amount of air he expels and the speed at which he does so. The amount of air expelled by maximal voluntary effort is the *vital capacity*. In normal subjects three-quarters of this air is expelled within the first

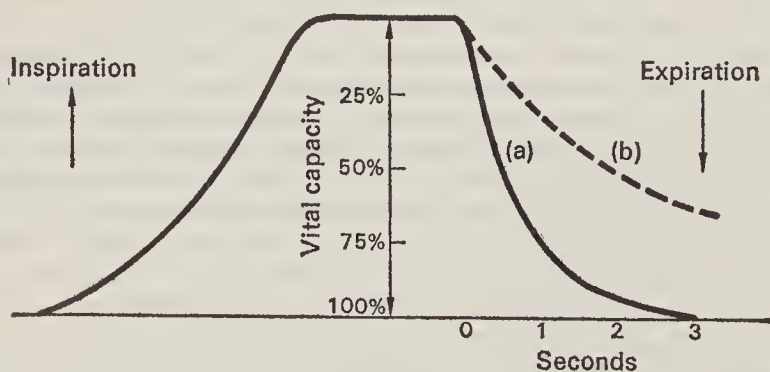


Fig. 39. The expiratory spirogram. (a) Normal. (b) In obstructive airways disease.

second and all within about 3 seconds. Thus the normal curve is a steep one. In diseases such as asthma, bronchitis and emphysema there is obstruction to the flow of air out of the lungs, owing to collapse or constriction of the airways, or intraluminal obstruction, owing to mucus or oedema, so that the curve is flattened (Fig. 39b). On scrutiny of the curves, this flattening is apparent, but a numerical value can be given to the degree of flattening by recording the volume expired in one second. This volume (*forced expiratory volume* at 1 second, or FEV_1) is then expressed as a ratio of the *vital capacity* (VC). This ratio (FEV_1/VC) is called the $FEV\%$ and should exceed 70% in healthy individuals under 60 years of age. If the percentage is below this figure, the patient may be described as suffering from *obstructive airways disease*. The test may be repeated after the use of a bronchodilator aerosol, and if the FEV_1 improves, the *airways obstruction* is said to be *reversible*. Broadly speaking, in asthma and bronchitis the obstruction is partly reversible by bronchodilators, whereas in emphysema this is not so. However, the distinction between these conditions is not as clear as might be supposed. Alternatively the maximum expiratory flow rate can be measured by means of a peak flow meter. This highly portable instrument (Fig. 40) measures directly the expiratory flow rate, which is reduced in airways obstruction.

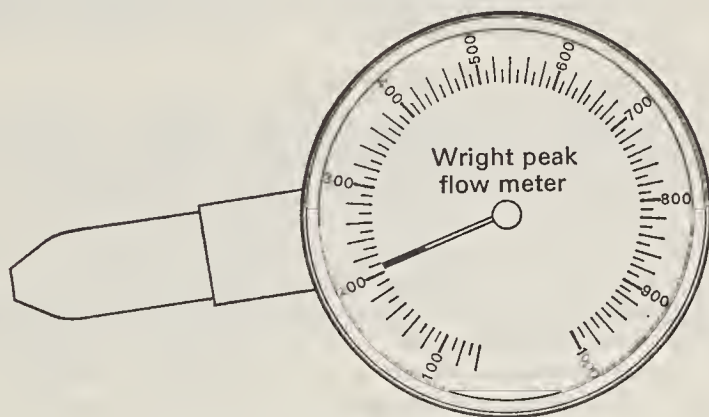


Fig. 40. A Wright peak flow meter.

Diffusion studies

Having entered the lungs, air is normally fairly evenly distributed throughout them. This distribution may be quite uneven in disease, but complex apparatus is needed to detect this.

Oxygen and carbon dioxide must pass freely between alveoli and pulmonary capillaries. The passage of these gases occurs largely by

diffusion. Carbon dioxide diffuses very readily and rarely presents a problem; but in certain conditions, notably the *interstitial fibroses*, and some pulmonary infiltrations, such as *sarcoidosis*, the diffusion of oxygen may be limited. Unequal *distribution* of inspired gas in relation to *perfusion* of the pulmonary capillaries may also cause abnormal oxygenation of the blood. The *diffusing capacity* or *transfer factor* of the lungs is measured by using *carbon monoxide* as a marker gas and the uptake of this gas is measured either during a single period of breath holding or by a steady-state technique. A reduced *diffusing capacity* or *transfer factor* can cause reduced oxygenation of the arterial blood, particularly on exercise.

The blood gases

The overall function of the lungs is to ventilate the blood adequately—that is to say, to maintain limits needed for normal tissue metabolism. In *respiratory failure* these physiological limits are exceeded. In particular, carbon dioxide is inadequately eliminated from the body and so its level in the blood rises in the same way as the blood urea rises in renal failure. If the PCO_2 is elevated above 45 mmHg we can say that the patient is in respiratory failure due to *alveolar hypoventilation*. If the PCO_2 rises further, clinical features of carbon dioxide retention appear and urgent treatment is required. In some instances, repeated estimation of PCO_2 may provide a valuable guide as to the efficacy of treatment which is given.

Direct measurement of the PCO_2 in arterial blood cannot always be made. Fortunately an alternative method, requiring neither arterial blood nor complex electrodes, has been introduced by Campbell and Howell. In principle this involves the patient rebreathing into a bag until the gas mixture in the bag is in equilibrium with alveolar air and hence indirectly with arterial blood. The final gas mixture in the bag is then analysed for carbon dioxide, using a simple apparatus.

The nomenclature of these measurements is complex, and reference should be made elsewhere for a fuller explanation. Normally the *arterial oxygen saturation* is above 97%, the *carbon dioxide tension* (PCO_2) is 37–43 mmHg and the pH 7.38–7.42. The concept of PCO_2 is an important one, as it is the *tension* or *partial pressure* of a gas that determines its diffusion between tissues or alveoli and systemic or pulmonary capillaries. Methods of measuring oxygen *tension* in blood have recently been developed and are replacing the measurements of *saturation* previously made.

Other tests

Two other simple measurements that may be useful are the *minute volume* and the *maximum voluntary ventilation*.

The *minute volume* may be calculated by multiplying the *tidal volume* (the volume of each breath) by the *respiratory rate*. It is usually measured by means of a spirometer or by collecting expired air into a bag. In either case breathing through a mouthpiece into a system presenting some resistance to air flow may cause the subject to increase his respiration. This effect may be diminished by using a low-resistance portable Wright's respirometer. The *minute volume* only measures air entering or leaving the respiratory tract at the mouth. Because the conducting airways (trachea, bronchi and bronchioles) act as a respiratory 'dead space' where no gas exchange occurs, the *alveolar ventilation* is always less than the *minute volume* (though proportional to it). Direct measurements of 'dead space' are not often made, and the adequacy of alveolar ventilation can be determined by estimation of the PCO_2 .

In the *maximum voluntary ventilation* test the subject breathes as hard and as fast as he can into a spirometer for a 15-second period. The volume of air he breathes during this period is multiplied by 4, to convert it to litres/minute. This test is dependent on several factors: (a) the full cooperation of the patient; (b) the neuromuscular function of the muscles of respiration; and (c) the presence or absence of airways obstruction. It is therefore not a test of any *single* aspect of lung function. It may serve as a guide as to what the patient is capable of achieving by voluntary effort and will be affected by neurological disease such as myasthenia gravis as well as by disease of the lung.

SKIN TESTS

Skin tests can be valuable in the diagnosis of chest disease. Allergic asthma is associated with Type 1 hypersensitivity and immediate skin reactions to allergens such as grass pollen and house dust mite. Delayed hypersensitivity (Type 4) is shown by Mantoux tests used to detect the presence of tuberculous infection (previous or recent). The Kveim test is relatively specific for sarcoidosis. Here an intradermal injection of antigen is made and the site of the lesion is biopsied for histological examination six weeks later.

8

THE SKIN

Colour and Pigmentation — Haemorrhages — Eruptions — Palpation — Examination for Parasites

For the examination of the skin and its appendages, the patient should be stripped as completely as circumstances permit and should be examined by daylight.

COLOUR AND PIGMENTATION

First notice the colour of the skin. The normal colour is very variable, some persons having a fresh complexion, and others, though quite healthy, a pale one. Pallor is also often seen in a variety of illnesses. It may be seen temporarily in haemorrhage, shock and intense emotion. Anaemic persons are often pale, but not all pale persons are anaemic. The colour of the mucous membrane of the eyelids and mouth is a better indication of anaemia than is the colour of the skin. *Undue redness* is seen in overheating, extreme exertion, sunburn, some fevers, in many of the exanthemata and in skin disease. *Cyanosis* is a bluish or purplish tint, which may be more or less generalized or limited to one or more extremities. It is due to the presence of an excess of reduced haemoglobin resulting from impaired oxygenation or circulation of the blood (p. 27). It is important to note that *methaemoglobinaemia* may produce a blue tint which is less bright and more leaden than cyanosis. Methaemoglobinaemia may be due to poisoning by aniline or nitrobenzene, or to drugs such as phenacetin, sulphanilamide or dapsone.

Jaundice varies from the 'subicteric', 'lemon-yellow', or 'daffodil' tints seen in pernicious anaemia and acholuric jaundice, to various shades of yellow, orange or dark olive green in obstructive jaundice. Jaundice must be distinguished from the yellowness of carotinaemia, due to the presence of an excess of lipid-soluble yellow pigments in the plasma. Carotene does not stain the conjunctivae, which jaundice does. Slight degrees of jaundice cannot be seen in artificial light.

Normal skin contains varying amounts of brown *pigment*. A congenital absence of pigment in the skin which is generalized is known as *albinism*; if it is localized it is known as *piebaldism*. Alternating patches of white and darkly pigmented skin are seen in leucomelanoderma or

PLATE I



Tetany



Gout



Rheumatoid arthritis



Heberden's nodes



Koilonychia



Clubbing

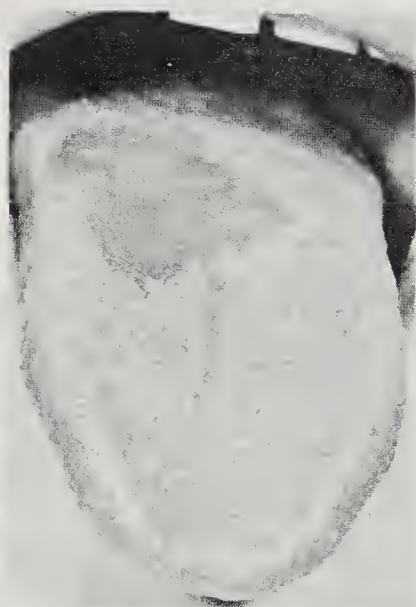
PLATE II



Black hairy tongue



Geographical tongue



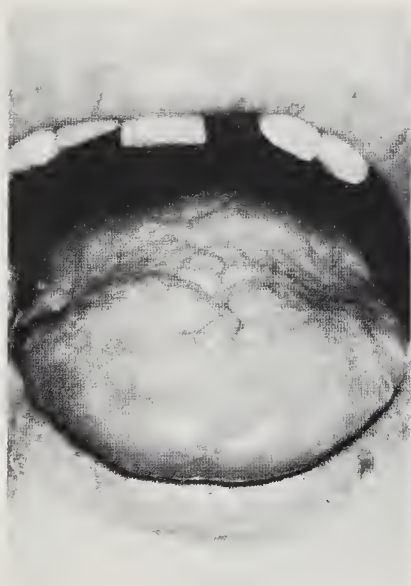
False geographical tongue

THE TONGUE

PLATE III



Congenital fissures



Chronic superficial glossitis



Median rhomboid glossitis

THE TONGUE

PLATE IV



Occlusion of inferior
vena cava



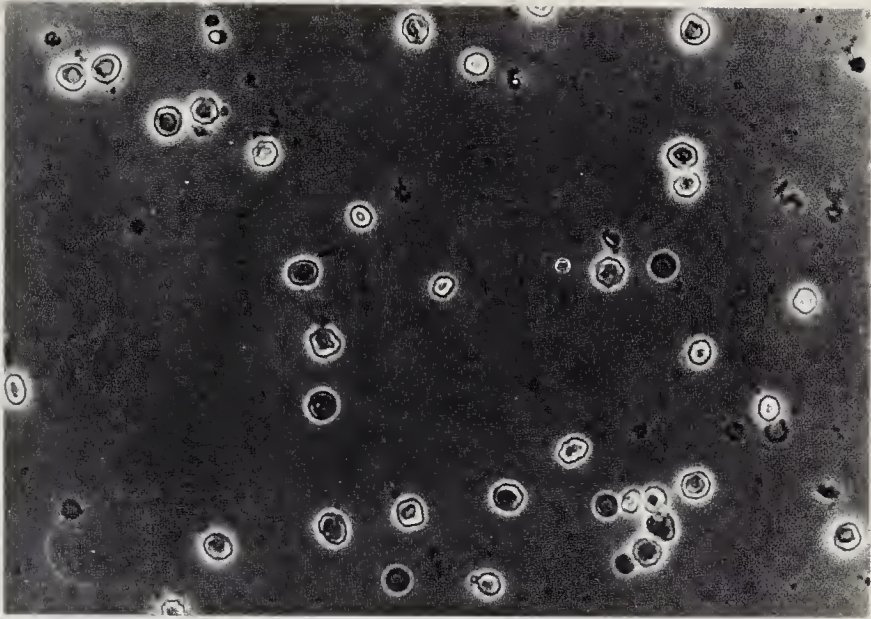
Venous anastomosis in
portal obstruction



Arterial anastomosis on shoulders
in coarctation of aorta

COLLATERAL CIRCULATIONS

PLATE V



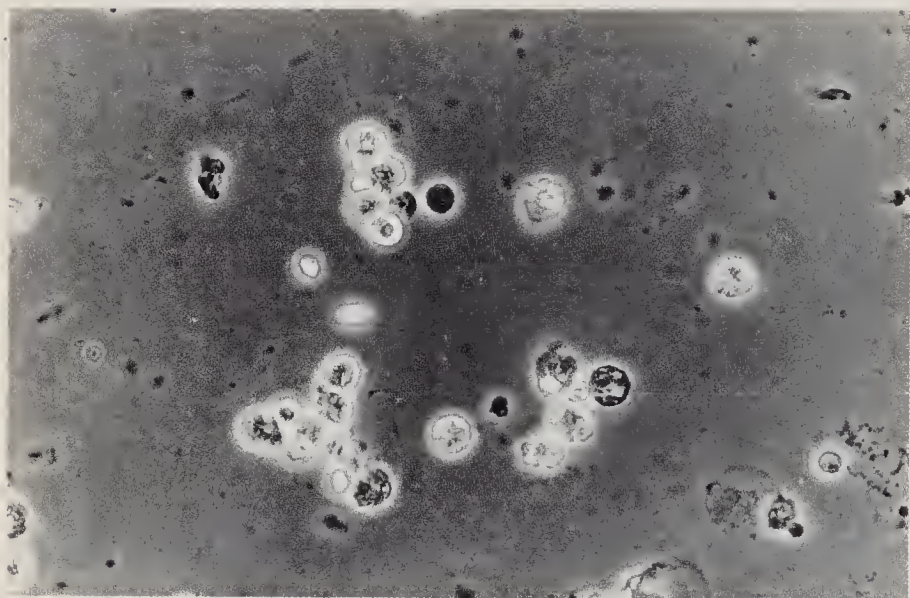
Erythrocytes

URINARY SEDIMENT

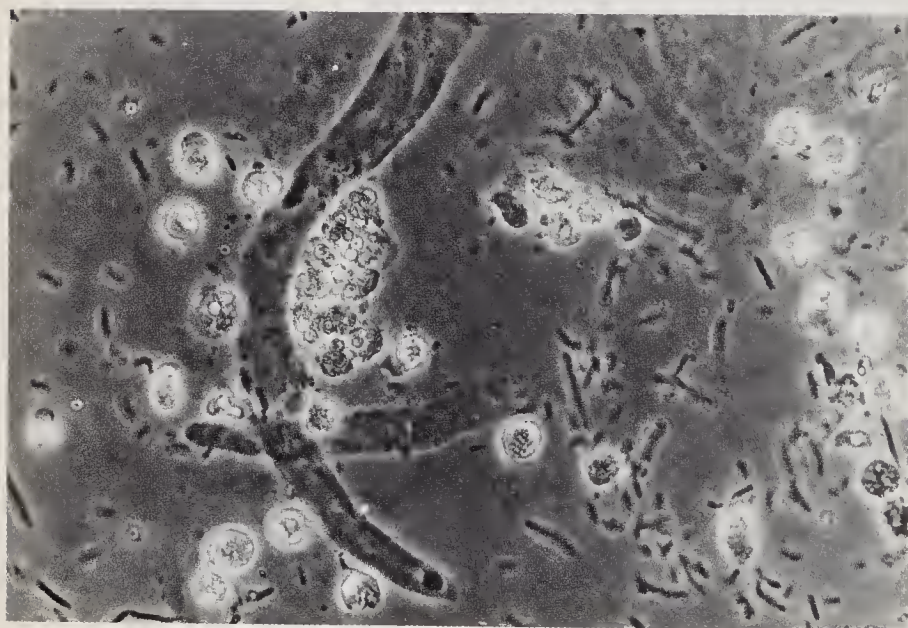
Plates V, VI and VII illustrate the urinary sediment as seen by phase contrast microscopy. Readers using ordinary microscopy must not expect to see casts and cells so clearly defined

Plates V, VI and VII reproduced by kind permission of the publishers from: Edwin S. Spencer and Ib Pederson (1971) Hand Atlas of the Urinary Sediment. Copenhagen: Munksgaard

PLATE VI



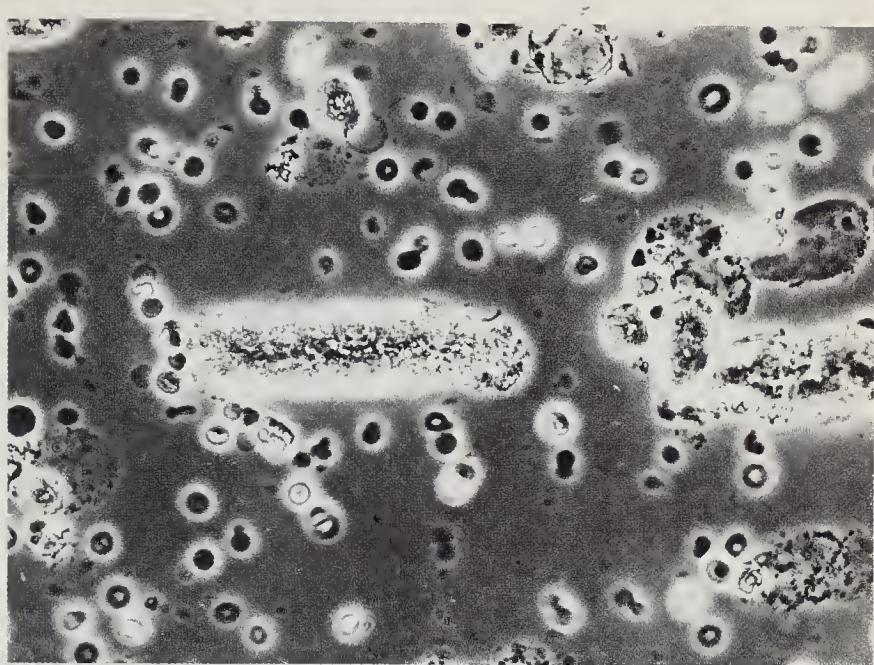
Leucocytes



Hyaline casts, leucocytes and bacteria

URINARY SEDIMENT

PLATE VII



Granular casts
URINARY SEDIMENT

PLATE VIII

INTESTINAL PARASITES

- 1 *Entamoeba histolytica*. Fully developed four-nucleated cyst, containing chromatid bodies, as seen in saline preparations. $\times 1500$
- 2 *Entamoeba histolytica*. Four-nucleated cyst as seen in iodine preparation. $\times 1500$
- 3 *Entamoeba histolytica*. Active form, containing included red blood cells, as seen in saline preparations. $\times 1500$
- 4 *Iodamoeba bütschlii*. Cyst, as seen in saline preparations. Note the unstained glycogen vacuole. $\times 1500$
- 5 *Entamoeba coli*. Fully developed eight-nucleated cyst, as seen in saline preparations. $\times 1500$
- 6 *Entamoeba coli*. Eight-nucleated cyst stained by Lugol's iodine solution. $\times 1500$
- 7 *Entamoeba coli*. Active form, as seen in saline preparations. $\times 1500$
- 8 *Iodamoeba bütschlii*. Cyst stained by Lugol's iodine solution. $\times 1500$
- 9 *Giardia lamblia*. Cyst form, stained by Heidenhain's haematoxylin. $\times 1500$
- 10 *Giardia lamblia*. Active form, stained by Heidenhain's haematoxylin. $\times 1500$
- 11 *Trichomonas hominis*. Stained by Giemsa's method. $\times 1500$
- 12 *Isospora belli* (*I. hominis*). Undeveloped oocyst as passed in human faeces. $\times 500$
- 13 *Balantidium coli*. Active form stained by Heidenhain's haematoxylin. $\times 350$
- 14 Ova of *Ankylostoma duodenale* (hookworm). $\times 500$
- 15 Ova of *Enterobius vermicularis* (threadworm). $\times 500$
- 16 Ova of *Taenia solium* and *T. saginata* (tapeworms). $\times 500$
- 17 Ova of *Trichuris trichiura* (whipworm). $\times 500$
- 18 Ova of *Ascaris lumbricoides* (roundworm). $\times 500$
- 19 Ova of *Schistosoma haematobium*. $\times 300$
- 20 Ova of *Schistosoma japonicum*. $\times 300$
- 21 Ova of *Schistosoma mansoni*. $\times 300$

All magnifications approximate

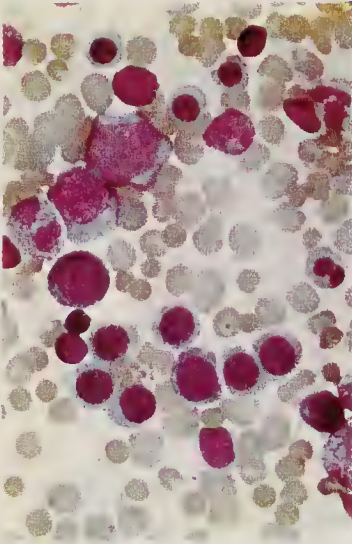
Drawings by W. Cooper

PLATE VIII

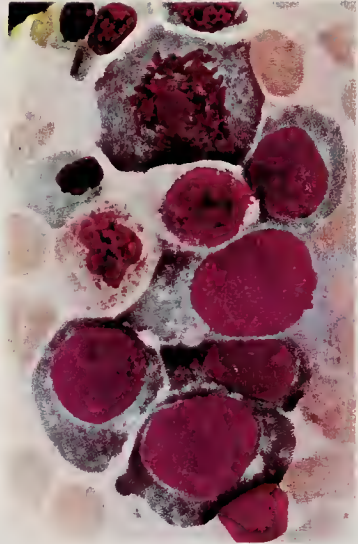


INTESTINAL PARASITES

PLATE IX

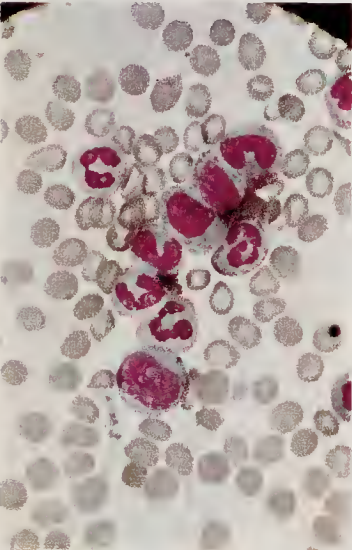


Normoblasts

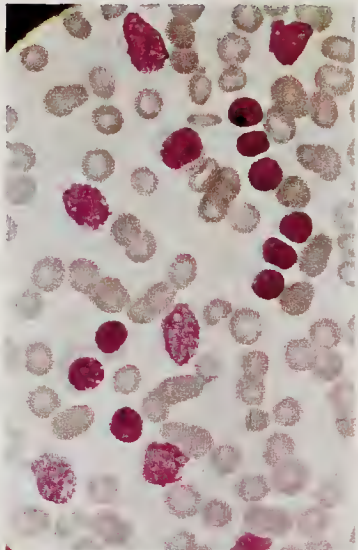


Megaloblasts, showing early, intermediate and late mitosis

BONE MARROW CELLS



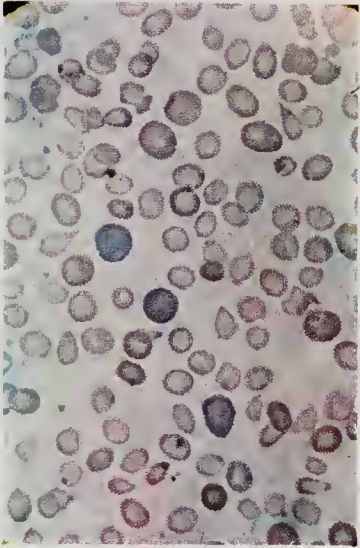
Chronic *myeloid*. Mature polymorphs, band forms, meta-myelocytes with indented nuclei, myelocytes with larger nuclei and granular cytoplasm, one larger myeloblast containing nucleoli



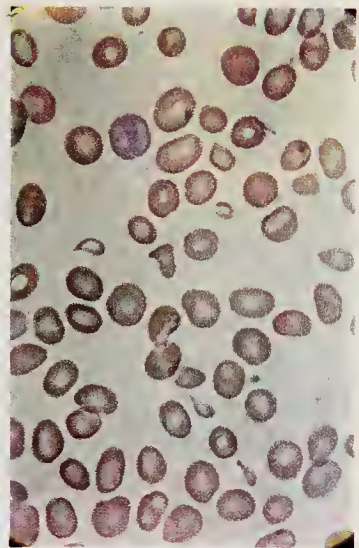
Chronic *lymphatic*. Large numbers of morphologically normal small lymphocytes; bare nuclei also present

LEUKAEMIA

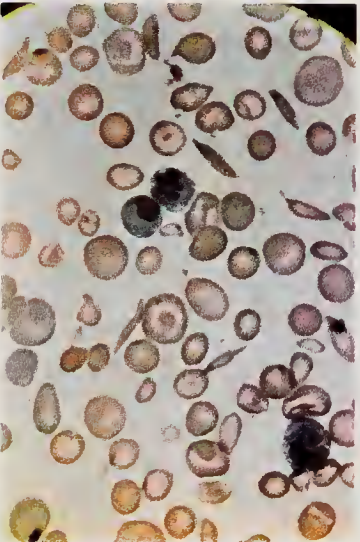
PLATE X



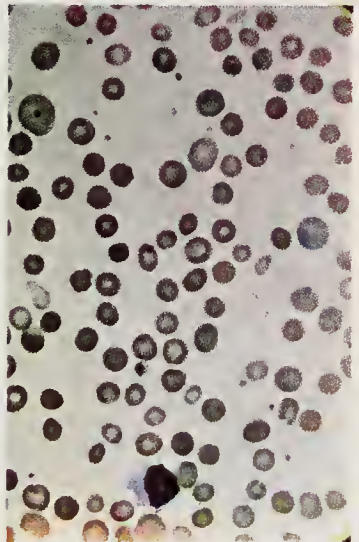
Hypochromic, due to blood loss. Mixed population of hypochromic microcytes, some normochromic cells of normal size, some polychromatic macrocytes



Pernicious. Anisocytosis, poikilosis and a number of macrocytic cells



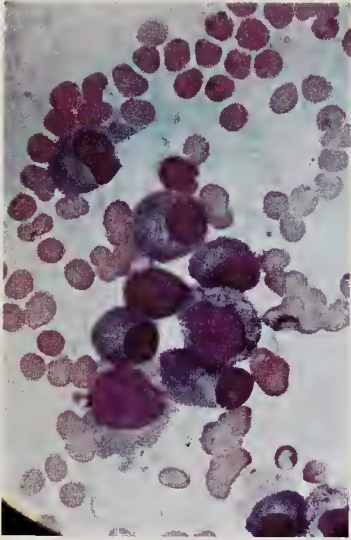
Sickle cell. Numerous target cells and some sickle cells. Hypochromic microcytes. Two normoblasts



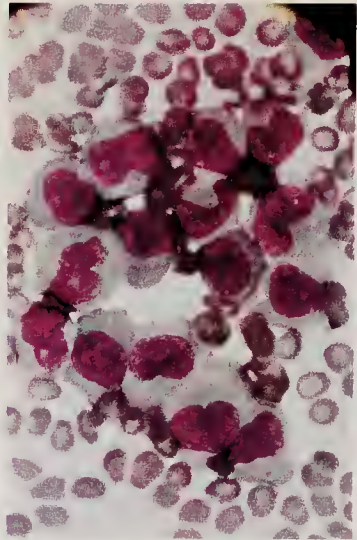
Hereditary spherocytosis. Some spherocytic red cells—smaller diameter and deeply staining centre. Some polychromatic macrocytes

THE ANAEMIAS

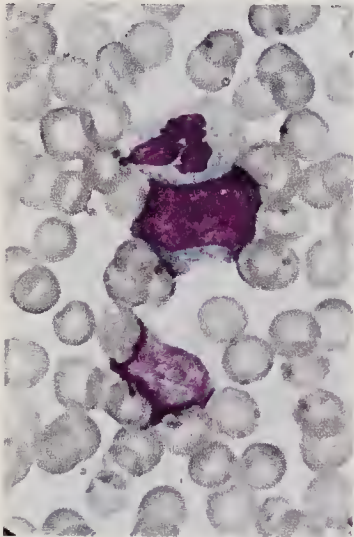
PLATE XI



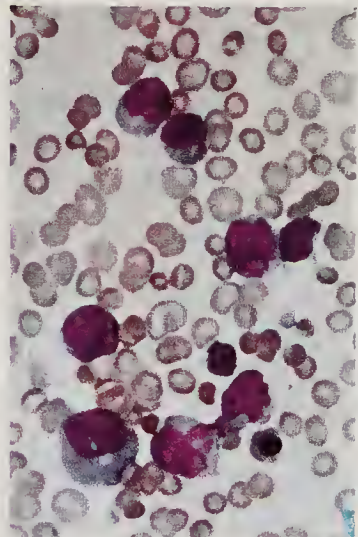
Bone marrow in myelomatosis.
Well-differentiated plasma cells



Blood cells in acute leukaemia.
Monocytic type



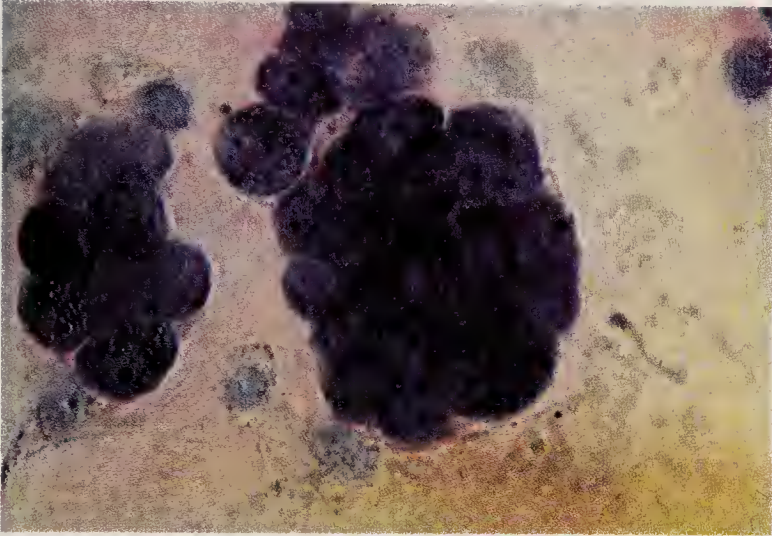
Peripheral blood in glandular fever. Normal lymphocyte below and basophilic atypical lymphocyte in the centre



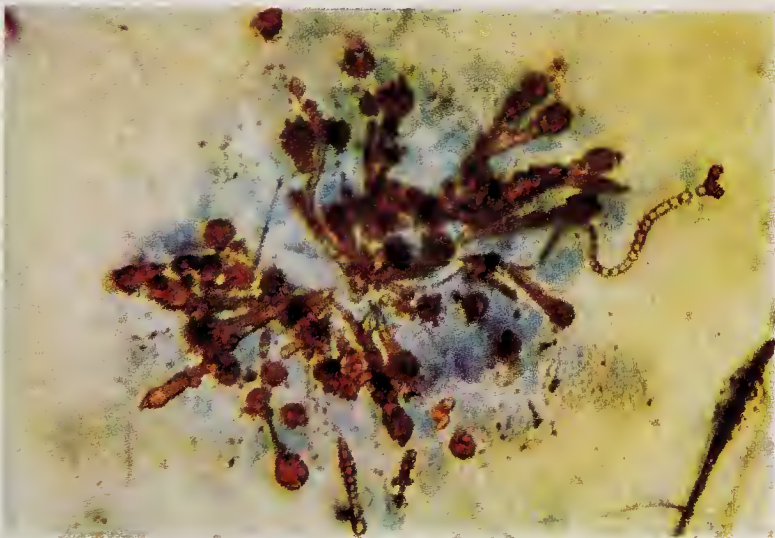
Peripheral blood in acute leukaemia. Atypical blast cells with nucleoli

MISCELLANEOUS BLOOD CELL DISORDERS

PLATE XII



Carcinoma cells



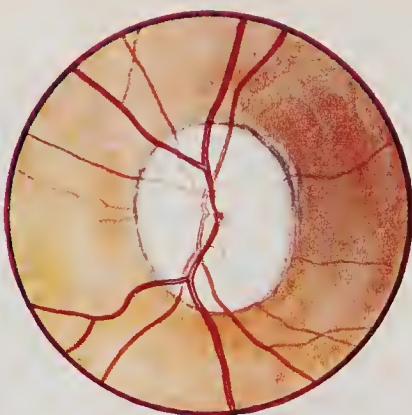
Asbestos bodies

SPUTUM

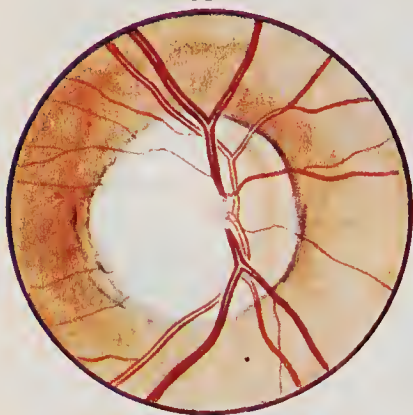
PLATE XIII



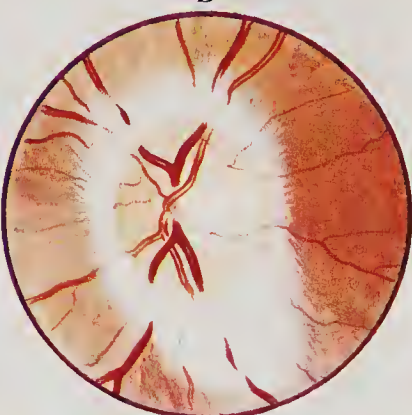
A



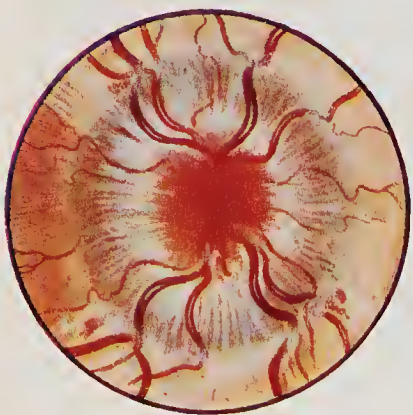
B



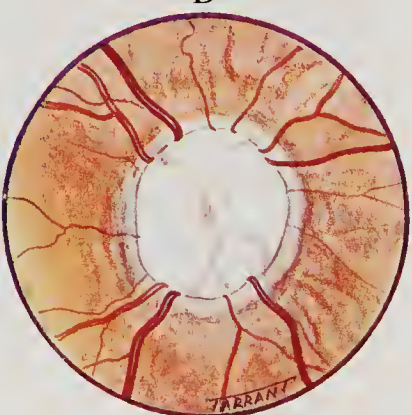
C



D



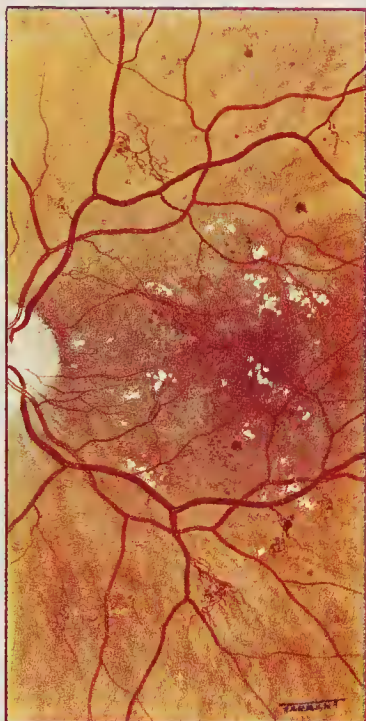
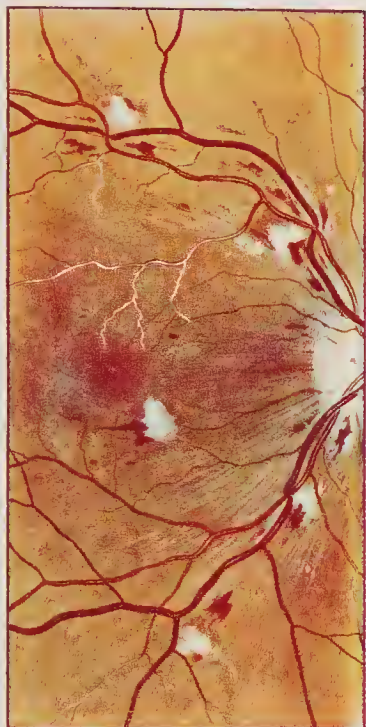
E



F

FUNDUS OF THE EYE

PLATE XIV



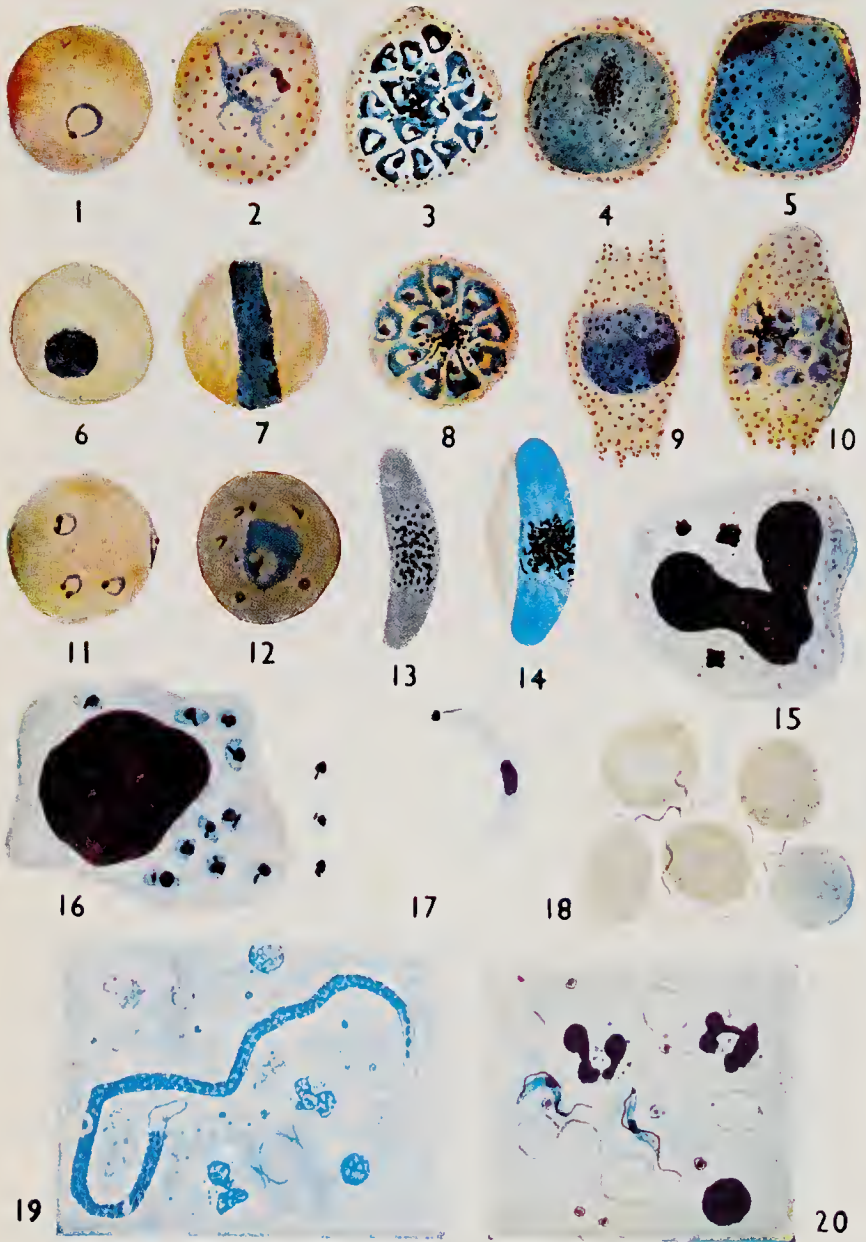
Hypertensive. The arteries are irregular in calibre and show 'silver-wiring'. Arteriovenous nipping is present. Characteristic 'flame-shaped' haemorrhages and 'cotton wool' exudates can be seen

Diabetic. Micro-aneurysms (tiny red dots), round haemorrhages, waxy exudates and areas of new vessel formation are characteristic of this condition. In many patients hypertensive retinopathy is also present

RETINOPATHY

- A *Normal optic disc*
- B *Optic atrophy.* The disc is white and the number of small vessels crossing its edge is reduced
- C *Myopic crescent.* A crescent or ring of exposed white sclera, seen particularly in high myopia
- D *Opaque nerve fibres.* The characteristic appearance of medullated nerve fibres
- E *Papilloedema.* A red swollen optic disc with dilated veins. The retinal vessels bend sharply as they dip down from swollen disc to surrounding retina. A similar appearance occurs in papillitis
- F *Glaucomatous cupping.* A form of optic atrophy due to raised intraocular pressure. The floor of the cup is depressed below the level of the surrounding retina

PLATE XV



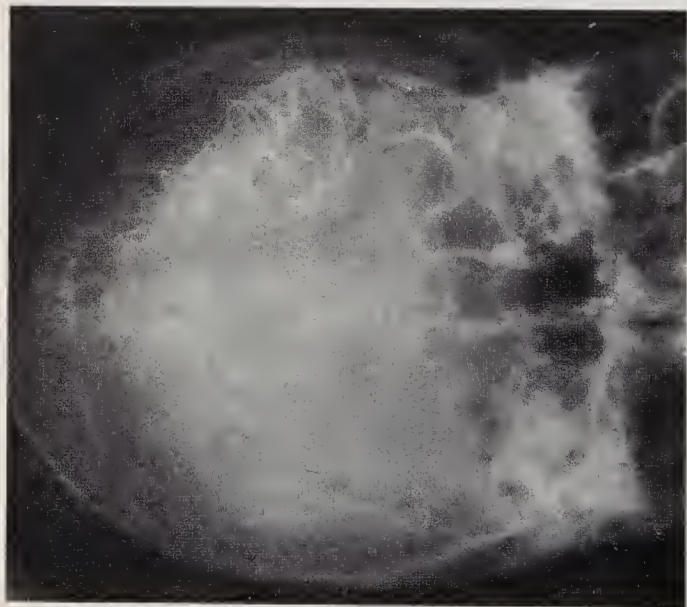
PARASITES OF THE BLOOD

PLATE XV

PARASITES OF THE BLOOD

- 1 *Plasmodium vivax*. Ring stage. $\times 2000$
- 2 *Plasmodium vivax*. Amoeboïd form. $\times 2000$
- 3 *Plasmodium vivax*. Fully developed schizont. $\times 2000$
- 4 *Plasmodium vivax*. Male gametocyte. $\times 2000$
- 5 *Plasmodium vivax*. Female gametocyte. $\times 2000$
- 6 *Plasmodium malariae*. 'Compact' form. $\times 2000$
- 7 *Plasmodium malariae*. 'Band' form. $\times 2000$
- 8 *Plasmodium malariae*. Fully developed schizont. $\times 2000$
- 9 *Plasmodium ovale*. Female gametocyte. $\times 2000$
- 10 *Plasmodium ovale*. Fully developed schizont. $\times 2000$
- 11 *Plasmodium falciparum*. Red blood corpuscles containing various types of young rings. $\times 2000$
- 12 *Plasmodium falciparum*. 'Old' ring, showing altered staining reaction and Maurer's dots. $\times 2000$
- 13 *Plasmodium falciparum*. Male gametocyte or crescent. $\times 2000$
- 14 *Plasmodium falciparum*. Female gametocyte or crescent. $\times 2000$
- 15 *Plasmodium falciparum*. Pigment in polymorphonuclear leucocyte. $\times 2000$
- 16 *Leishmania donovani* from a spleen smear. Some lying free and others within the cytoplasm of an endothelial cell. $\times 2000$
- 17 *Trypanosoma cruzi*. Adult form as seen occasionally in the blood of patients suffering from Chagas's disease. $\times 2000$
- 18 *Borrelia recurrentis*. $\times 2000$
- 19 *Microfilaria loa loa*. $\times 600$
- 20 *Trypanosoma rhodesiense* as seen in a thick blood film of patients suffering from trypanosomiasis. $\times 1000$

Drawings by W. Cooper



Subdural haematoma shown on a left carotid angiogram. Note the midline displacement of the pericallosal artery to the right. Peripheral branches of the left middle cerebral artery are displaced away from the inner table of the skull



Thrombosis of the right internal carotid artery, with total occlusion at its origin. Note the dilated external carotid artery and branches

PLATE XVII



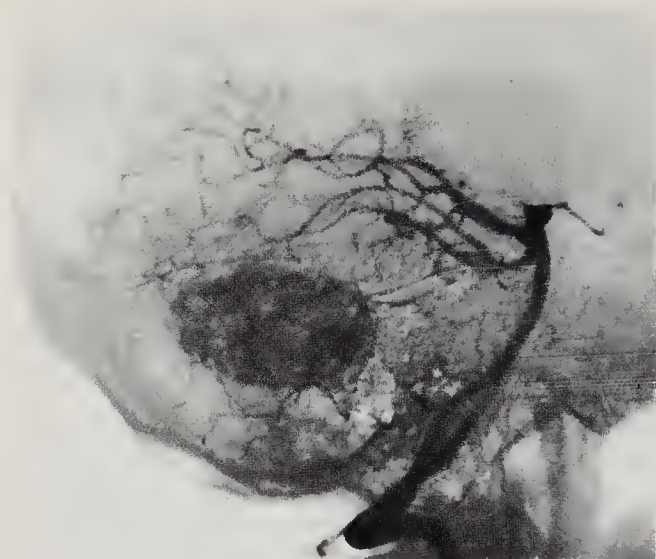
Right acoustic neuroma. Tomogram through brain stem seen outlined by gas in the ambient cisterns. Right internal auditory canal widened with soft tissue mass (★) in right cerebellopontine angle cistern



Pituitary tumour with suprasellar extension (★). Recurrence of basophil adenoma. Metallic object in pituitary fossa is clip previously applied to pituitary stalk. *Inset.* Normal appearance after surgical removal of the tumour in the same patient

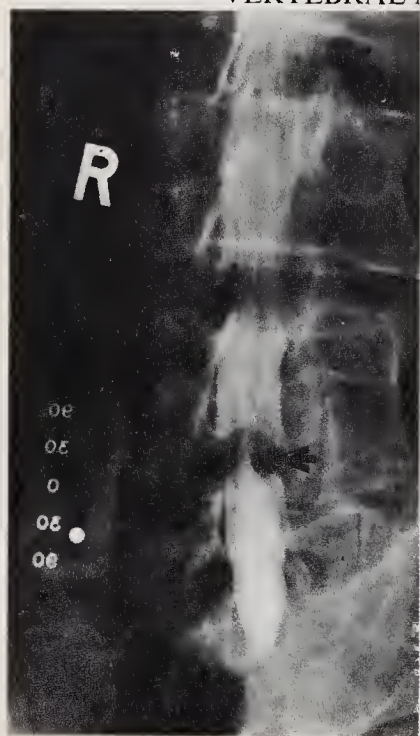
PNEUMOENCEPHALOGRAPHY

PLATE XVIII



Haemangioma of a cerebellar hemisphere. Pathological circulation outlines the vascular tumour

VERTEBRAL ANGIOGRAPHY



Lumbar disc prolapse, with narrowing of the thecal sack on the left side at L4-5 level

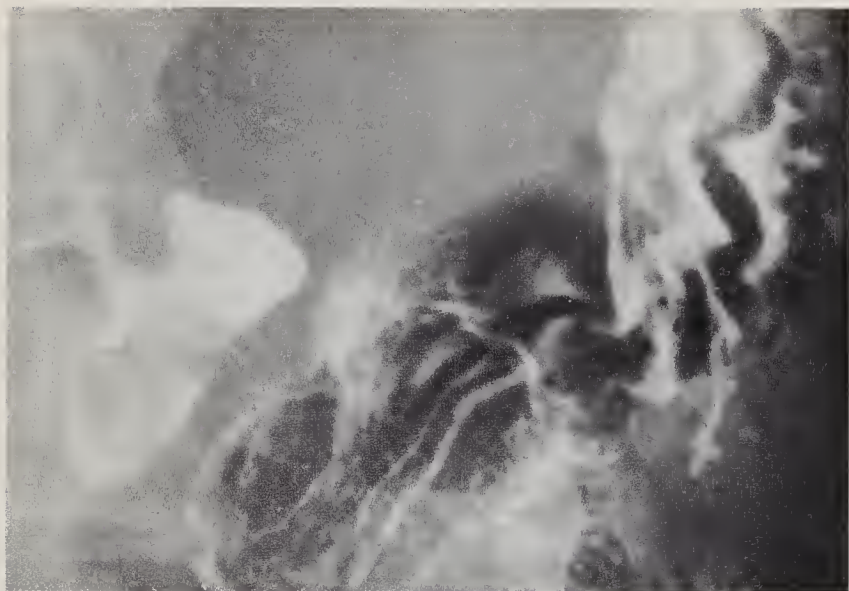
LUMBAR MYELOGRAPHY

PLATE XIX

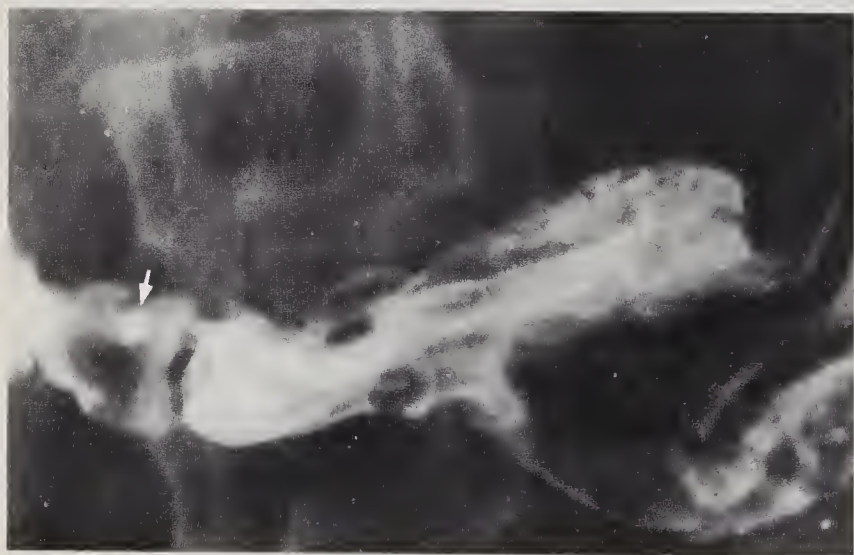


Infiltrating carcinoma of the distal end of the stomach
BARIUM MEAL

PLATE XX



Trefoil deformity of chronic duodenal ulceration



Duodenal ulcer *en face*

BARIUM MEALS

PLATE XXI



Acute ulcerative colitis affecting the whole colon
BARIUM ENEMA

PLATE XXII



Chronic ulcerative colitis with polypoid carcinoma at the
splenic flexure

BARIUM ENEMA

vitiligo. A pale skin due to diminished pigment is also characteristic of *hypopituitarism* and *hypogonadism*. Increased pigmentation may be racial, due to sunburn or connected with various diseases. In *Addison's disease* there is a brown or dark brown pigmentation, affecting exposed parts, parts pressed on parts normally pigmented such as the axillae and, very characteristically, the mucous membranes. That of the lips and mouth should always be examined and may exhibit dark bluish-black areas that have been compared with the stains produced by sucking a pen. Note, however, that mucous membrane pigmentation is a normal finding in a substantial proportion of negroids. More or less generalized pigmentation may also be seen in *haemochromatosis*, where it has a peculiar bronze colour with a metallic sheen; in *chronic arsenic poisoning*, where it is finely dappled, and affects covered more than exposed parts; in *argyria*, where the deposition of silver in the skin produces a diffuse slatey-grey hue; and occasionally in the cachexia of advanced malignant disease. In *pregnancy* there may be pigmentation of the nipples and their areolae, of the linea alba and sometimes a mask-like pigmentation of the face (chloasma). Localized pigmentation may be seen in *hyperthyroidism*, *pellagra*, *rheumatoid arthritis* and a variety of chronic wasting diseases. Localized pigmentation is also seen in scars of various kinds, particularly those due to X-irradiation therapy and those following varicose ulcers of the legs. *Phtheiriasis* or *vagabond's disease*, a pigmentation due to chronic infestation with lice, is now rarely seen. *Erythema ab igne*, a coarsely mottled pigmentation of the legs of women who habitually sit too near a fire, is, however, common.

HAEMORRHAGES

Haemorrhages into the skin occur in various forms and in various conditions. If less than 1 mm in diameter they should be referred to as *petechiae*; if from 2 to 5 mm in diameter as *purpuric spots*; and if larger as *ecchymoses*. If the haemorrhage is large enough to produce an elevation of the skin, it is referred to as a *haematoma*.

Petechiae and purpuric spots do not disappear when they are pressed on by a glass slide or lens, which serves at once to distinguish them from erythematous spots, such as the rose spots of typhoid fever, and from telangiectases, which consist of a small collection of dilated skin vessels. They must also not be confused with *senile haemangiomas* (de Morgan's spots) or cherry spots, which are common and have no pathological significance.

ERUPTIONS

Next, one should seek the presence of any eruption. If present, inquiry should be made on the lines laid down on p. 15. The exact situation and

extent of the eruption should be noted and whether it is symmetrical or confined to one side only. One should then pass to a description of the minute characters of the eruption. In order to do this, it must be remembered that every cutaneous eruption consists of a primary lesion, to which secondary lesions may or may not be superadded.

Primary lesions

Macules (spots). Any abnormal change in the colour of the skin confined to a limited area. Always note whether or not they fade on pressure. The rose spots of typhoid fever, for example, fade on pressure, whilst those due to haemorrhages into the skin do not.

Papules. Solid projections above the surface, which are not larger than a pea. The term *nodule* is applied to any solid projection from the skin which is larger than a pea, but not larger than a cherry. Anything larger than that is called a *tumour*. Always note whether the top of the papule is rounded as in some forms of eczema, pointed as in acne or flattened as in lichen. As regards the base, observe whether it infiltrates the skin widely or not. The wider the infiltration, the more extensive and severe the inflammation.

Vesicles. Elevations of the horny layer of epidermis by transparent or milky fluid, which are not larger than a pea. If larger than this, they should be described as *bullae* or *blebs*. Always note whether or not there is an area of redness around the base of a vesicle, for such redness indicates that the vesicle is planted upon an inflamed base—a fact which may be of diagnostic value.

Pustules. Small elevations of the skin containing pus. Observe whether there is much infiltration around them or not.

Weals. Slightly elevated portions of the skin, the centre of which is paler than the periphery.

Having stated which of these primary lesions it is that composes the eruption, one should next note whether the lesions are isolated (*discrete*), or whether they run together (*confluent*). It must also be remembered that an eruption may be made up of more than one kind of primary lesion. Thus, papules may be mingled with pustules, or pustules with vesicles, and so on.

Secondary lesions

Next look for *secondary lesions*. These are either produced mechanically or are the result of changes which take place in the primary lesion in the course of its growth or decline. The commonest secondary lesions of

mechanical production are *excoriations* due to scratching, and *fissures*—deep cracks going down to or into the corium, and produced by the stretching of the skin after it has become inelastic owing to thickening of any kind. Fissures are often very painful.

The following are the secondary lesions produced by changes in those which are primary:

Desquamation. If the primary lesion is a dry one (macules or papules) a scaling off of epidermic cells occurs and the eruption is then said to be '*scaly*'.

In moist lesions (vesicles, pustules, bullae) the epidermic cells become glued together by the dried fluid and a *scab* or *crust* forms. The scab may be serous, purulent, haemorrhagic or sebaceous according to the nature of the contents of the primary lesions.

Infiltration may occur around the primary lesions, leading to a leathery feeling in the skin. This is usually the result of prolonged chronic inflammation.

Pigmentation may occur around the primary lesions. This also is usually due to prolonged inflammation.

Ulceration. Caused by breaking-down of the primary lesions and destruction of a part of the true skin.

The points to note in describing an ulcer are: (*a*) the nature of the floor of the ulcer and the granulations covering it; (*b*) the character of the edge—smooth, raised, undermined, etc.; (*c*) the discharge, whether serous, purulent, watery, fetid, etc.; and (*d*) the character of the surrounding skin, whether indurated, pigmented, etc. It is also important to examine the lymph nodes that drain the area of the ulcer.

Scar formation. This only occurs where the true skin has been involved, i.e. where there has been an ulcer or an equivalent injury. Describe the scar, noting especially whether it is thin or thick, freely movable or adherent to the deeper tissues, pale or livid, pitted or not, surrounded by a zone of pigmentation or not.

PALPATION

Proceed now to the palpation of the skin. Pass the hand gently over it, pinching it up between the forefinger and thumb, and note the following points:

Is it smooth or rough, thin or thick, dry or moist? If there is any visible sweating, note whether it is general or local. The *elasticity* of the skin

should be investigated. If a fold of healthy skin is pinched up, it immediately flattens itself out again when released. Sometimes, however, it only does so very slowly, remaining for a considerable time in a creased condition. This may be of little or no significance in old persons with loose inelastic skins, but may be an important sign of dehydration in conditions associated with prolonged vomiting and diarrhoea.

The conditions of the subcutaneous tissue should be investigated. The presence of *oedema* is usually recognized by the fact that if the skin is pressed with the finger, especially over a hard body such as a bone, a pit is left which persists for some little time. In some cases, no pitting can be produced, especially when the oedema is of very long standing. The best place to look for slight degrees of oedema in cardiac disease is behind the malleoli of the tibia and fibula in patients who are ambulant, and over the sacrum in those who are confined to bed. The pressure of the finger should be maintained for 20–30 seconds, or small degrees of oedema will be overlooked. Pitting is absent in oedema due to lymphatic obstruction, where the skin is usually thickened and tough.

Subcutaneous emphysema gives rise on palpation to a characteristic crackling sensation. It starts in, and is usually confined to, the neighbourhood of the air passages or air-containing organs. In rare cases it may be due to infection with gas gangrene organisms.

EXAMINATION FOR PARASITES

Microscopical examination of the skin and its appendages is useful in the diagnosis of some *parasitic diseases*, of which the following are the chief:

Scabies or itch

This is due to the *Acarus (Sarcoptes) scabiei*. The female *Acarus* is larger than the male and forms burrows in the skin, in which the eggs are deposited. These burrows should be looked for between the fingers and on the inner aspects of the wrists. They are recognized with the naked eye as little short dark lines terminating a sort of shining spot of skin. The eggs lie in the dark line, the insect in the shining spot. It may be picked out by means of a flat surgical needle passed along the black line to the clear spot. The use of a lens aids the operation—which is by no means invariably successful—and makes possible the recognition of the insect. The latter may be placed on a slide under the microscope for more detailed examination.

Pediculosis

Three varieties of pediculosis occur—*Pediculus capitis* on the head, *P. corporis* on the trunk, *P. pubis* on the pubic and axillary hairs. The eggs

or 'nits' of *P. capitis* are stuck on the hairs. From their position on the hairs one can judge roughly of the duration of the condition, for they are fixed at first near the root of the hair, and are then carried up with the latter in its growth. The higher up the nits are, therefore, the longer the pediculi have been present. *P. corporis* should be looked for in the seams of the clothes, especially where the latter come into close contact with the skin—e.g. over the shoulders. The bites of the parasite produce haemorrhagic spots, each with a dark centre and a paler areola. Marks of scratching should always be looked for on parts accessible to the patient's nails.

P. corporis is the longest of the three, *P. pubis* is shortest, and *P. capitis* is between the two in size. *P. pubis* is also distinguished from the others by being yellowish-brown in colour. *P. capitis* and *P. corporis* are both greyish in colour, though the latter varies considerably with the colour of the skin of its host. The shape of the thorax and abdomen forms a distinguishing character between these varieties and *P. pubis*.

Fungus infections

Fungus may grow in the skin, nails, or hair and cause disease (ringworm).

Skin. Between the toes, on the soles of the feet and in the groins are the commonest sites. The lesions may be scaly and vesicular areas tending to spread in a ring form and healing in the centre; scaly erythematous plaques with festooned margins; areas of hyperkeratosis on the parts of the skin which have a thick horny layer (palms and soles); or macerated, dead-white offensive-smelling epithelium in the intertriginous areas such as the toe clefts.

Nails. Discoloration, deformity, hypertrophy and abnormal brittleness may result from fungus infection.

Hair. Ringworm of the scalp is most common in children. It presents as round or oval areas of baldness covered with short, broken-off, lustreless hair stumps. These hair stumps usually give a bright green fluorescence when exposed to long-wave ultraviolet light (UVL filtered through Wood's glass).

Microscopical examination

Scales from the active edge of a lesion are scraped off lightly with a scalpel or the roofs of vesicles are snipped off with scissors. The material is placed in a drop of 10–20% aqueous potassium hydroxide solution on a microscope slide, covered with a cover slip and left for 30 minutes to clear. It is then examined with the 8 mm or 4 mm objective, using low

illumination. The mycelium is recognized as branching refractile threads which boldly transgress the outlines of the squamous cells (mycelium which respects the cells' outlines is 'mosaic fungus', an appearance probably produced by intercellular lipoid). Nails are examined in much the same way, but as nail is harder and denser it is necessary to break up the snippings and shavings into small fragments. These are either heated in potassium hydroxide or are left to clear in it overnight before being examined. A scalp lesion is cleaned with 70% alcohol or with 1% cetrimide and infected stumps and scales are removed by scraping with a scalpel. The hairs are cleaned in potassium hydroxide in the same way as skin scales. Examination under the microscope reveals spores on the outside of the hair roots, and mycelium inside the hair substance. The species of fungus responsible may be established by culture on Sabouraud's glucose-agar, or on beerwort-agar medium.

9

THE NERVOUS SYSTEM

Anatomy and Physiology — Mental Functions — Speech — The Cranial Nerves — Motor Functions — Sensation — Signs of Meningeal Irritability — Special Investigations — Routine Examination of the Nervous System

The aim of a neurological examination is to determine the site and nature of disease of the nervous system. In simplified form it is also an essential part of any routine clinical examination. Clinical examination of the nervous system should be thought of as a technique for precisely delineating the patient's disability in physiological and anatomical terms. It gives information about the condition at the time of the examination but gives no clue as to the natural history, nor therefore, to the pathogenesis of the disorder. For the latter precise history taking is of particular importance since it alone enables the evolution of the disease to be studied.

A detailed neurological examination is an ordeal for ill patients and a test of concentration and cooperation for those in good general health. Care should be taken not to fatigue the patient unduly. Overlong examination may defeat its own ends, especially when sensation is being investigated, by leading to variable and incongruous findings. It may therefore be necessary to conduct the examination in more than one session.

The order of examination outlined in this chapter need not be rigidly adhered to. For example, if a patient is complaining of sciatic pain, it is appropriate to begin with the examination of the lower limbs and lumbar spine. An abbreviated scheme for routine use is set out at the end of the chapter. Observation of the patient's ordinary activity, for example the way he walks into the room and undresses for examination, is often helpful in deciding how to begin the examination.

ANATOMY AND PHYSIOLOGY

The dominant hemisphere is that which plays the major part in control of a person's abstract activities, especially of language. The left hemisphere is dominant in almost all right-handed people and in most left-handed people.

The motor system

The lower motor neurone

Muscular movement depends ultimately on the integrity of the lower motor neurones, which connect striped muscles with the central nervous system. The lower motor neurones consist of the anterior horn cells, homologous cells in the brain stem and their efferent nerve fibres, which pass via the anterior spinal nerve roots and peripheral nerves to the muscles.

If this final common pathway is interrupted at any point, *weakness, fasciculation, muscle wasting, loss of tendon reflexes and hypotonia* occur. These are the cardinal signs of a *lower motor neurone lesion*.

Although various reflex movements operate at a spinal level, the initiation of voluntary movements and the maintenance of posture and muscle tone depend on impulses arising from higher centres. These impulses can only reach the muscles if the final common path is intact. These higher centres consist of the corticospinal and extrapyramidal systems and the cerebellum.

The corticospinal (pyramidal) system

This system consists of the pathways which directly link the pyramidal cells in the fifth layer of the motor cortex with the lower motor neurones in the brain stem and spinal cord. The fibres concerned are gathered together in the corticospinal tracts. The corticospinal tracts, however, also contain fibres which arise from the postcentral cortex and subcortical structures. The motor area of the cortex occupies the anterior wall of the central sulcus (Rolandic fissure) and the adjacent parts of the precentral gyrus. There is localization of function in the motor cortex, different parts of the opposite side of the body being separately represented. Those parts of the body which carry out the most skilled movements, for example the fingers and thumb, have the largest areas of representation. The areas for tongue, jaw and facial movements lie lowest in the motor cortex, those for the arm, trunk and leg following successively as the motor area ascends on to the medial aspect of the hemisphere (Fig. 41).

The fibres of the corticospinal tracts pass downward from their cells of origin into the internal capsule, occupying the anterior two-thirds of the posterior limb. Here the order of representation of the body is face, shoulder, elbow, hand, trunk and lower limb from before backwards. The corticospinal fibres then descend to occupy the middle three-fifths of the peduncles of the mid-brain in the same order. Passing through the pons, the tract becomes broken into scattered bundles by the transverse pontine fibres and nuclei pontis. In the upper part of the medulla the fibres join to form the pyramids, which appear as well marked protuberances on the anterior aspect of the brain stem. In the lower part of the medulla the majority of the corticospinal fibres decussate with those of the opposite side and pass posteriorly to run down the spinal cord in the lateral columns as the crossed corticospinal tracts. A smaller number of fibres do not decussate, but continue downwards in the anterior columns as the direct corticospinal tracts, which decussate at seg-

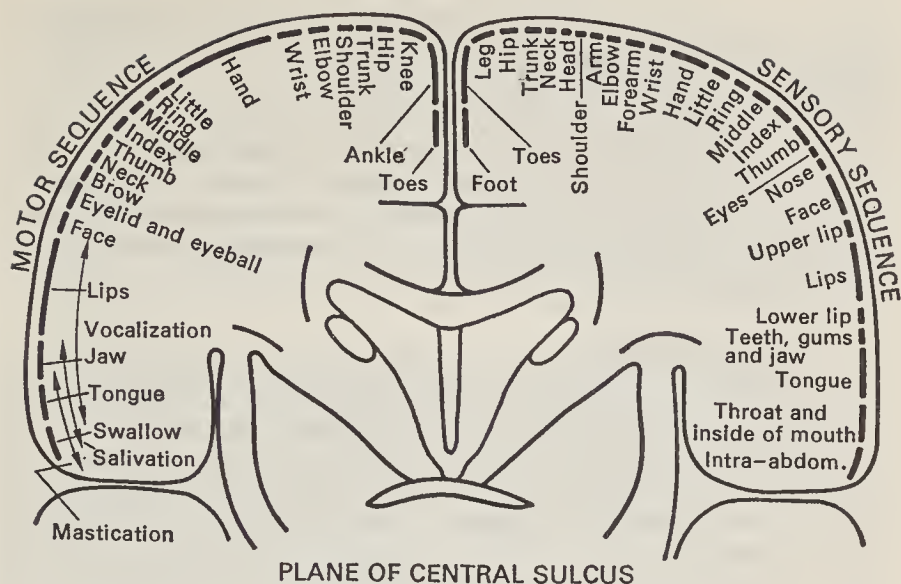


Fig. 41. Rasmussen and Penfield's diagram of localization in the motor (left) and sensory (right) cortex.

mental levels in the anterior commissure. A few uncrossed fibres descend in the crossed corticospinal tract of the same side, ending in the anterior horns of the same side. The corticospinal fibres terminate at different levels in the grey matter of the brain stem or in the anterior horns of the spinal cord.

The corticospinal system is concerned with the initiation of voluntary and skilled motor acts, particularly of fine distal movements. Contrary to former teaching the paralysis resulting from lesions affecting corticospinal fibres alone is limited to impairment of fine and rapid distal movements of the digits, as in picking up a small object. The more familiar occurrence of widespread paralysis of one side of the body (*hemiplegia*) or of a single limb (*monoplegia*) is usually the result of a more extensive lesion affecting extrapyramidal or other subcortical structures in addition to the corticospinal fibres themselves. Even in cases of dense hemiplegia movements of the head and trunk (axial movements) usually escape altogether. The nervous pathways for such postural movements are predominantly under subcortical control and are represented bilaterally.

Since, in clinical practice, most lesions of the corticospinal system also damage neighbouring extrapyramidal nuclei and pathways the distinctions drawn above are rarely of clinical importance. All such cases are loosely grouped as 'corticospinal lesions'. The classical signs of such a lesion (an *upper motor neurone lesion*) are *weakness*, which

predominantly affects fine, distal movements, hip flexion and shoulder abduction, *spasticity*, *increased tendon reflexes*, and an *extensor plantar response* (p. 245).

When the corticospinal system is suddenly damaged or destroyed, as by haemorrhage or injury, there is a temporary depressant effect on the anterior horn cells (neuronal shock). Paralysis is accompanied at first by loss of muscle tone and absent or reduced tendon reflexes. The characteristic hypertonia and increased reflexes of a corticospinal lesion appear after a few hours or days.

The extrapyramidal system

This term is applied to those parts of the nervous system, excluding the motor cortex and corticospinal pathways, which are concerned with movement and posture. The system includes the basal ganglia, the subthalamic nuclei, the substantia nigra, the red nuclei and other structures in the brain stem. The connections of these extrapyramidal centres are complex and include fibres from the cerebral cortex and the thalamus. There are no direct pathways from the basal ganglia to the spinal cord; the connections with the lower motor neurones are indirect, via several paths arising in the brain stem. These include the dentatorubrospinal, reticulospinal, vestibulospinal and olivospinal tracts.

The extrapyramidal system, which must clearly be considered in close relation with the corticospinal system, is important in the control of posture and in the initiation of movement, especially those movements which affect postural mechanisms such as sitting, standing, turning over in the lying position, walking and running. Complex volitional movements, such as reaching for an object, require both postural adjustments and fine distal movements, which are themselves broadly speaking under corticospinal control.

Diseases affecting the extrapyramidal system are characterized by difficulty in initiating voluntary movement, by impairment of orienting and balancing reflexes, by alterations in muscle tone and by the appearance of involuntary movements. Muscle power is rarely weakened.

The cerebellum

The cerebellum receives afferent fibres from the spinal cord, vestibular system, basal ganglia and cerebral cortex. It influences the lower motor neurone mainly through its connections, via the thalamus, with the basal ganglia and cerebral cortex.

Lesions of the cerebellum cause muscular hypotonia and incoordination (ataxia). Paralysis is not a feature of cerebellar disease. Lesions of the cerebellar vermis cause a characteristic ataxia of the trunk, so that the patient has difficulty sitting up or standing. In such patients there may be little or no incoordination of the limbs.

The sensory system

Sensory input reaches the nervous system from specialized receptors and free nerve endings in the skin and superficial tissues; from other receptors, such as

muscle spindles, Golgi tendon organs, Pacinian corpuscles and free nerve endings in muscles; and from other specialized receptors in the joints. All afferent fibres enter the central nervous system through the posterior root ganglia and the posterior roots. Disease of these 'first sensory neurones' may thus affect all modalities of sensation. It must be remembered, however, that much sensory input is concerned with the reflex control of posture and movement and as such does not reach consciousness or is not consciously perceived. Thus the conscious recognition of posture and position of a limb is dependent on input from cutaneous and joint receptors. Muscle spindles and tendon organs also seem to play some part in this sensation (kinaesthesia) although clearly their major role is in the control of voluntary and reflex movements.

After they have entered the spinal cord, the various sensory fibres are rearranged and grouped into other systems. The majority of these afferent fibres terminate in the grey matter of the posterior horn at or near the level at which they enter and from this grey matter the secondary sensory tracts take origin. Some cross immediately, or within a few segments, to the opposite lateral and anterior columns of the cord and in it ascend to the brain stem. Impulses from which the sensations of pain and temperature are derived ascend in the *lateral spinothalamic tract*, the fibres from the lower part of the body being placed laterally. Other peripheral fibres, however, do not synapse in the grey matter of the spinal cord, but ascend in the ipsilateral posterior columns: these posterior column fibres carry impulses upon which depend the appreciation of position, of movement, of size, shape, discrimination and texture, and of vibration (which should be regarded only as touch rapidly applied). The medial of the two posterior columns, the *fasciculus gracilis*, contains fibres originating in the lower part of the body, whereas the lateral, the *fasciculus cuneatus*, carries fibres predominantly from the upper limbs. It should be noted, therefore, that somatotopic lamination of fibres in the posterior columns is the converse of that in the lateral spinothalamic tracts.

At any level of the spinal cord, therefore, there are two major groups of sensory fibres conveying sensory information towards the brain: one in the anterior and lateral columns carrying pain and temperature from the opposite half of the body, and a second in the posterior column, conveying the appreciation of posture, weight, size, shape and other qualities of sensation from the same side of the body. A unilateral lesion of the spinal cord, therefore, results in loss of pain and thermal sensibility below the level of the lesion on the opposite side of the body, while on the side of the lesion there is, in addition to spastic paralysis, disturbance of the sense of position and of movement and loss of recognition of weight, size, shape, touch and vibration. This group of clinical signs is called the *Brown-Séquard syndrome*.

At the upper end of the spinal cord the posterior column fibres terminate in the gracile and cuneate nuclei. The fibres of the secondary sensory neurone originate in these nuclei and immediately cross to the opposite side of the medulla in the sensory decussation. In the medulla, therefore, *all* sensory impulses are carried in secondary tracts situated on the opposite side to that from which they arise. But even here all do not run in a single pathway: spinothalamic fibres pass through the lateral part of the medulla, while those afferent impulses carried by the posterior columns enter the medial lemniscus.

Higher in the brain stem the two sensory pathways are joined by the secondary sensory fibres from sensory cranial nerve nuclei. Finally the fibres of the medial lemniscus and spinothalamic tract terminate in the thalamus. All secondary sensory fibres synapse in the thalamus. From this level a third system of sensory fibres conveys sensory impressions through the internal capsule to the cerebral cortex.

The description of the major sensory pathways given above is sufficient for the practical work of diagnosis in the neurological clinic, but it is clearly a gross oversimplification of a complex subject. There are, for example, many afferent pathways other than the two described. These two pathways themselves are not modality specific in the sense that certain fibres 'carry' vibration sense or other forms of sensation: but rather the interaction of different patterns of impulses in different sized fibres of differing conduction velocity determines the sensation perceived at the highest levels of the nervous system. The importance of spinal mechanisms in the control of the flow of afferent information has recently been emphasized by Melzack and Wall in their 'gate control' theory of sensation, to which the reader is referred for a full discussion of this topic. This work has emphasized, furthermore, the pre-eminent role of the dorsal columns in exploratory, movement-directed behaviour, rather than in the passive reception of sensory input.

The spinal cord

The cord extends caudally to the interspace between the 12th thoracic and 1st lumbar spines; the thecal membranes are continued down as far as the body of the 2nd sacral vertebra. The cervical enlargement reaches to the 7th cervical spine. Its largest part is at the level of the 5th and 6th cervical vertebrae. The lumbar segments lie opposite the 10th and 11th thoracic spines and the next interspinous space.

Since the cord ends at the level of the lower border of the first lumbar vertebra, spinal segments do not correspond with the vertebrae overlying them. To determine which spinal segment is related to a given vertebral body:

For the cervical vertebrae, add 1.

For thoracic 1-6, add 2.

For thoracic 7-9, add 3.

The 10th thoracic arch overlies lumbar 1 and 2 segments.

The 11th thoracic arch overlies lumbar 3 and 4 segments.

The 12th thoracic arch overlies lumbar 5.

The 1st lumbar arch overlies the sacral and coccygeal segments. In the lower dorsal region the tip of a spinous process is on a level with the body of the vertebra below.

The spinal cord is made up of segments from each of which a pair of anterior (motor) and posterior (sensory) nerve roots arises. The *myotomes* and *dermatomes* supplied by the pairs of nerve roots are shown in Tables 5 and 6 and in Fig. 42. It is important to remember these,

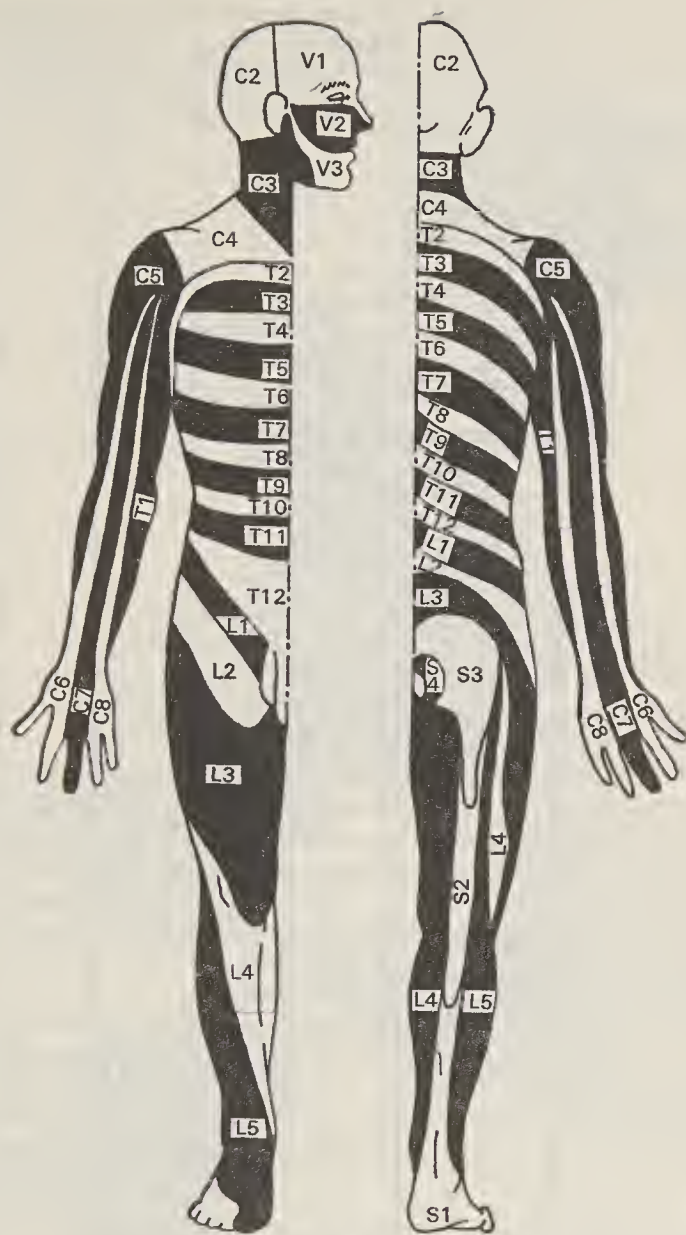


Fig. 42. The cutaneous areas supplied by sensory roots. Minor variations are common.

TABLE 5. SEGMENTAL INNERVATION OF THE MUSCLES
OF THE UPPER LIMB

CERVICAL SEGMENTS					DORSAL
	5	6	7	8	1
SHOULDER	SUPRASPINATUS				
	TERES MIN.				
	DELTOID				
	INFRASPINATUS				
	SUBSCAPULARIS				
ARM		TERES MAJOR			
	BICEPS				
FOREARM		TRICEPS			
		BRACHIO-RADIALIS			
		SUPINATOR			
		EXTENSOR CARPI RADIAL.			
		PRONATOR TERES			
		FLEXOR CARPI RADIAL.			
		FLEXOR POLLIC. LONG.			
		ABDUCT. POLL. LONG.			
		EXTENS. POLL. BREV.			
		EXTENS. POLL. LONG.			
		EXTENS. DIGITOR.			
		EXTENS. INDICIS.			
		EXTENS. CARPI. ULN.			
		EXTENS. DIGITOR. MIN.			
		FLEXOR DIGITOR. SUBLIMIS			
		FLEXOR DIGITOR. PROFUND.			
		PRONATOR QUADRAT.			
		FLEX. CARPI. ULN.			
		PALMARIS LONG.			
HAND		ABDUCTOR POLL. BREV.			
		FLEXOR POLL. BREV.			
		OPPONENS POLL.			
			FLEXOR DIGIT. MIN.		
			OPPONENS DIGIT. MIN.		
			ADDUCT. POLL.		
			PALMARIS BREV.		
			ABDUCTOR DIGIT. MIN.		
			LUMBRICALES		
			INTEROSSEI		

TABLE 6. SEGMENTAL INNERVATION OF THE MUSCLES
OF THE LOWER LIMB

	DI2	L1	L2	L3	L4	L5	S1	S2
HIP	ILIO-PSOAS				TENSOR FASCIÆ			
					GLUTEUS MEDIUS			
					GLUTEUS MINIMUS			
					QUADRATUS FEMORIS			
					GLUTEUS MAXIMUS			
THIGH					OBTURATOR INTERN.			
			SARTORIUS					
			ADDUCT. LONG.					
			QUADRICEPS					
			GRACILIS					
			ADDUCTOR BREVIS					
				OBTURATOR EXT.				
				ADDUCT. MAGN.				
				ADDUCT. MINIM.				
					SEMITENDINOSUS			
LEG					SEMI MEMBRANOSUS			
					BICEPS FEMORIS			
					TIBIALIS ANT.			
					EXTENS. HALL. LONG.			
					EXTENS. DIGIT. LONG.			
					SOLEUS			
					GASTROCNEMIUS			
					PERONEUS LONG.			
					PERONEUS BREV.			
					TIBIALIS POST.			
FOOT					FLEXOR DIGITOR. LONG.			
					FLEXOR HALLUC. LONG.			
					EXTENS. HALL. BREV.			
					EXTENS. DIGIT. BREVIS			
					INTRINSIC MUSCLES OF THE FOOT			
					INTEROSSEI			

since accurate knowledge of them enables quick and easy separation of nerve root and peripheral nerve lesions to be made by simple clinical examination. The sensory loss found after some common peripheral nerve lesions is shown in Figs 43 and 44. They should be carefully compared with the patterns of sensory loss shown in Fig. 42. Note that C8 and C6 sensory loss extends to the elbow on both ventral and dorsal surfaces of the forearm, but that ulnar and median nerve sensory loss predominantly affects the palmar surface of the hand and extends no further proximally than the wrist.

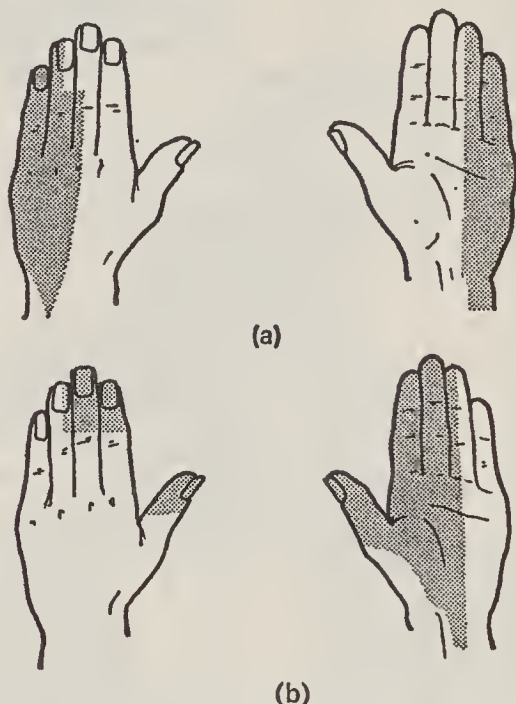


Fig. 43. Cutaneous sensory loss. (a) After division of the ulnar nerve above the elbow. (b) After division of the median nerve in the arm. These areas are subject to considerable variation.

Vascular supply of the brain and spinal cord

The *brain* is supplied by the internal carotid and vertebral arteries. Owing to the position of origin of the left common carotid, an embolus can enter it more easily than it can the artery of the opposite side. Embolic lesions are therefore more frequent in the left than in the right cerebral hemisphere.

The two *vertebral arteries* unite at the lower border of the pons to form the basilar, which runs up the middle of the anterior surface of the pons, dividing into the two posterior cerebrals. It gives off paramedian and short and long circumferential branches which supply the pons and parts of the mid-brain and cerebellum.

The *posterior cerebral* supplies the occipital lobe, the lower part of the temporal lobe and the uncus, the inner part of the crus and the corpus quadrigeminus, and the posterior part of the posterior limb of the internal capsule. Occlusion of this artery at its origin will therefore involve the visual cortex and the sensory fibres, but thrombosis often involves the calcarine branch and hence the visual cortex alone.

The *internal carotid* gives off the *anterior cerebral* artery, which curves round the anterior end of the corpus callosum, and is chiefly distributed to the inner surface of the cerebral hemisphere as far back as the parieto-occipital fissure. It also supplies the superior frontal gyrus, and gives a branch to the anterior part of the internal capsule and to the basal ganglia.

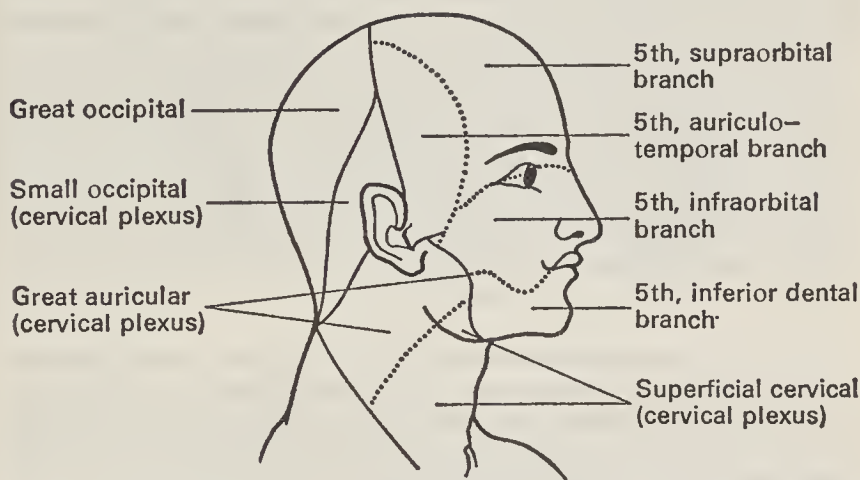


Fig. 44. The distribution of the sensory nerves of the head. Compare with the segmental distribution shown in Fig. 42.

The internal carotid is predominantly directed into the *middle cerebral artery*, which lies in the lateral sulcus (Sylvian fissure). An embolus which has found its way into the internal carotid, therefore, usually ends in the middle cerebral or one of its branches. The middle cerebral gives off cortical branches, which supply the motor area and the upper part of the parietal and temporal lobes. These branches anastomose freely with those of adjoining arteries so that occlusion of one of them may be largely compensated by the establishment of a collateral circulation. It also gives off *central branches* which penetrate into the brain substance and supply the white matter and the basal ganglia. There are two chief groups of these central arteries—an anterior group called the *lenticulostriate*, and a posterior group, the *lenticulo-optic*. The lenticulostriate are the source of hypertensive cerebral haemorrhage more often than are the lenticulo-optic. These central arteries also anastomose with one another.

Venous blood leaves the brain in the *venous sinuses*. Blood from the interior of the brain is chiefly returned by the cerebral veins, which end in the straight sinus.

Spinal arteries

The *anterior and posterior spinal arteries* arise from the vertebrals and travel downwards in the pia mater, the former in the anteromedian fissure and the two latter alongside the posterior nerve roots. Although they have a long and tortuous course, they do not diminish in size, being reinforced by radicular tributaries from the intercostal and lumbar arteries. The anterior spinal artery supplies most of the spinal cord, only the posterior parts of the posterior horns and columns being supplied by the posterior spinal arteries. Both anterior and posterior spinal arteries function as anastomotic vessels linking the radicular feeding vessels. Flow does not therefore occur in any particular direction in these vessels but varies, or may even reverse, in response to local factors such as posture and intra-abdominal and intrathoracic pressure variations.

The chief *veins of the spinal cord* are situated dorsally and ventrally in the middle line. Like the arteries, they communicate by radicular branches with the lumbar and intercostal veins, and empty into the vertebral veins. The blood in them flows upwards; hence in compression of the spinal cord, as by tumour or tuberculous abscess, there is venous engorgement below the level of pressure. This may very rarely result in a secondary, haemorrhagic, venous infarction of the spinal cord with paralysis and loss of sensibility below the level of the lesion. *However, if spinal compression is treated promptly, before cord infarction has occurred, recovery may occur.*

The formal and complete method of examining the nervous system is described in the following pages. The mental state and intellectual functions, including speech, are described first, followed by the methods for examination of the cranial nerves, and then the motor, sensory and reflex functions of the trunk and limbs.

MENTAL FUNCTIONS

It is important to analyse the patient's intellectual state early in the examination, even if the analysis is limited to a subjective opinion of personality, memory, education and abstractional ability formed during the process of history taking, since this opinion is helpful in the subsequent investigation of symptoms. For example, if the patient's memory is deficient, only a limited value can be attached to his account of his illness or the state of his previous health. Or if he is comatose or unable to understand speech, any attempt to investigate the state of his sensory functions is likely to be frustrated. In such cases, or in any case in which for any reason the patient's own history is incomplete, it is essential to obtain a history from the relatives or friends.

Appearance and behaviour

The patient's bearing or his actions when lying in bed are important. Note whether there is any disturbance of consciousness such as con-

fusion, stupor or coma. Is he unduly disturbed or apathetic, or in a state of agitation or terror? Is his attention easily held or fleeting? Does he show a reasonable degree of interest in his surroundings? How does he react to your approach and greeting? Is he well-groomed or unkempt? What is the condition of his hair, his beard, his nails and his hands? Any other features which strike one as unusual about his behaviour (e.g. facial tics or any inappropriate behaviour) should be noted.

Note whether his conversation flows easily or not: whether he is mute, answers only by monosyllables or is over-talkative. Do his remarks hold together as replies to questions? Do they show looseness of association? Do they show *flight of ideas* (a rushing stream of ideas with some connection) or the '*Knight's move*' in association (when one remark follows another with only indirect connection)? This name derives from chess, in which all pieces move on straight or diagonal lines, except the knight, whose move involves a change of direction. Do his replies to questions suggest *thought blockage*, or does he keep on repeating your question or his own remarks (*perseveration*)? He may use strange words (*neologisms*) or normal words strung together oddly (*word salad*).

Emotional state

It is important to note his mood. Is he happy or distressed? Is he happier than his condition would warrant (*elation* or *euphoria*) or filled with despair or dismay (*depression*)? Does his conversation lead you to feel that there is flattening of emotion, e.g. he speaks of family or financial success without pleasure or in an incongruous manner (he laughs after relating a misfortune or breaks into tears when given some pleasant news)? Is he able to enjoy anything? Does he feel that nothing is worth while? Does he feel fed up with life, or so fed up that he might as well end it (so that there is a risk of suicide)? Does the play of his features suggest that he enjoys a private world of his own, such as smiling or grimacing at odd times? Does he appear perplexed? Ask him whether persons or things seem as real as they once were, or whether they seem changed in some mysterious way (*depersonalization*). It is, however, unwise to ask leading questions about depersonalization, as neurotic patients will often respond affirmatively. Note whether the patient seems irritable or resentful, or whether he receives your words with suspicion.

Inquiries should be made about the patient's *sleep*. Does he sleep too much or too little? If too much, is any particular action likely to precipitate the hypersomnia? If too little, is the difficulty that of falling off to sleep, or of waking frequently, or waking early in the morning and being unable to go to sleep again? Where there is no physical cause for

the insomnia, such as pain, cough, asthma or the wearing of some uncomfortable plaster, the insomnia is likely to be due to some psychological disorder, e.g. the restlessness of mania, the early waking of depression, or the turmoil of the mind with difficulty in getting off to sleep in the anxiety states.

Inquire about *dreams*. These are frequent and sleep-disturbing in the anxiety states, whether the source of the anxiety is apparent or not. In these conditions sleep is not refreshing and the patient may complain of being as tired in the morning as at night.

Delusions and hallucinations

Delusions are false beliefs which continue to be held despite evidence to the contrary. Hallucinations are false impressions referred to the organs of special sense (hearing, seeing, smelling, etc.) for which no cause can be found and which the patient knows to be imagined or unreal events. The patient's conversation may have already indicated that these are present. Note their content and the patient's attitude when you express doubt about what seems so real to him.

Neither delusions nor hallucinations may be voiced spontaneously. They have then to be inquired for and the introduction of the subject may call for considerable tact. Is his mind or his body interfered with by others or by some physical agency, e.g. electricity, radio or TV, or poison? Has he felt that others talk about him or shun him? Are his relatives, neighbours and colleagues at work kind to him or difficult to get on with? Has he ideas of supernatural power or inordinate wealth or, conversely, does he feel that he is weakened physically, morally or financially? Has he feelings of guilt? Does he think that he caused his own illness?

Hallucinatory experiences may be carefully hidden. Auditory and visual hallucinations are commonest. Of these visual hallucinations are usually organic and, except in temporal lobe epilepsy, auditory hallucinations are usually due to inorganic psychoses such as schizophrenia. Does he hear or see anything unusual? Does he taste or smell what he has not expected? Hallucinations may only occur at certain times of the day or in certain places and this should be noted. Delusions which are secondary to the hallucinatory experiences must be noted, e.g. that 'the neighbours are against him, because his bedroom is filled with a gas, which he assumes the neighbours have engineered'. Notes should be made of any unusual actions upon the patient's part which have been prompted by delusions or hallucinatory ideas, e.g. ideas that his wife is in the pay of his enemies, or his clutching at small animals which he 'sees' crawling over his bed-clothes.

Orientation in place and time

Does the patient appreciate his surroundings and know where he is, or is he wholly ignorant, and does he try to explain his ignorance in some way such as *confabulation*? Can he tell the date approximately, if not perfectly correctly? Can he tell the approximate time without looking at a watch or clock? Does he know why he is in hospital?

Clouding of consciousness

It is important to recognize states of clouded consciousness and to be able to describe and define them to others, particularly in the context of patients with head injury or raised intracranial pressure who may gradually deteriorate during a period of clinical observation. *Coma* is a state in which the patient makes no psychologically meaningful response to external stimulus or to inner need. In *stupor* the patient, although inaccessible, does show some response, for instance, to painful stimuli. Above these deep levels lie various degrees of altered consciousness and lethargy, which may be accompanied by confusion. The questioner must be alert to observe any minor defects in the patient's capacity to grasp what is required of him and what has happened to him. Such defects will usually be manifest in the responses to tests for orientation, recent memory and appreciation of environment.

Memory

Inability to grasp and retain images and ideas is a marked feature in acute toxic-delirious reactions and in the subacute and chronic organic psychoses. In these cases recent events may have been registered but cannot be recalled, although later when recovered, the patient may be able to remember them. It is more often correct that, in such patients, events have not been registered.

The degree to which recent memory is lost is an index of the severity of organic brain disorder (not necessarily permanent). Inquire about the day of the week and of the month and the names of prominent public figures. Ask the patient to recall what he has read in the paper or seen on television. In formulating questions on these lines, regard should be paid to the patient's educational background and his likely personal interests. More subtle changes are discovered by seeing whether the patient can repeat seven digits forwards or five digits backwards. Bring up a subject discussed 3 minutes previously or give the patient a simple story or address to remember and note how much is remembered after a short interval.

General intelligence

It is usually necessary to ascertain the patient's general intelligence or how it has been affected by brain injury or disease. The standard which

he reached before leaving school, the character of his work and his work record give a rough-and-ready approximation. Frequent changes of job may indicate mental defect or personality disorder. Frequent changes after an accident or a serious illness with a previously good work record is suggestive of mental impairment.

Tests of memory as given above will indicate the more serious defects and these can be further exposed by tests of reasoning, more particularly where the tests show inability to criticize. Ask the patient to take sevens from a hundred (i.e. 100, 93, 86, 79 . . .), or to reverse in his mind's eye the hands of a clock. The absurdities test (e.g. 'What would be absurd if I told you I had three brothers, John, Fred and myself?') indicates grosser disability. A man with relatively low intelligence can give the months of the year parrot-fashion but is unable to say which month precedes May and which October, etc.

Tests in which the patient is asked the meaning of rare words or to interpret common proverbs or sayings are commonly used as an index of abstractional abilities. Judgement may similarly be assessed: 'What would you do if you saw a house on fire? Or a stamped addressed envelope lying in the road?' These questions often provoke concrete answers or a confused reply in the demented patient. Constructional and drawing tests may also be useful. These are briefly described on p. 258.

SPEECH

In considering speech disorders, it is essential to distinguish between defects of articulation and enunciation of speech (*dysarthria*) and those disturbances of the structure and organization of language itself, which we speak of as *aphasia*.

Dysarthria

Supposing that the patient is able to speak, one should note whether there is any abnormality in *articulation* apart from the variability inherent in various regional and other accents. The following are the chief abnormalities which may be present:

Stammering. This is a developmental disorder, more common in boys than girls. It is only very rarely due to organic brain disease.

Lalling or baby speech. Ask the patient to read something aloud. In lalling all the difficult consonants are dropped; he speaks like a baby. Lalling is usually the result of congenital or infantile deafness: very rarely it may be due to a congenital defect in the appreciation of the meaning of sounds—congenital auditory imperception.

Scanning or staccato speech (cerebellar dysarthria). The patient speaks slowly and deliberately, syllable by syllable, as if scanning a line of poetry. The normal prosodic rhythm of syllable, word and sentence production is lost and in extreme forms each syllable is given equal emphasis. Ask him to say 'artillery': he will pronounce it 'ar-til-ler-y'. This is the classical form of *cerebellar dysarthria*. It is important to recognize it in minor, less obvious forms since its presence indicates bilateral disease of the cerebellum or its brain stem connections.

Spastic dysarthria. The syllables are slurred together as in a state of intoxication. Thus 'British Constitution' becomes 'Brizh Conshishushon'. This form of dysarthria is due to bilateral lesions in the cortico-spinal fibres supplying the muscles of the face, larynx, tongue and respiration concerned with speech, i.e. the lesion is supranuclear to, or above the level of, these brain stem nuclei. It is therefore a manifestation of *pseudobulbar palsy*: the jaw jerk is invariably brisk.

Bulbar palsy, due to lower motor neurone lesions affecting the speech musculature, results in some non-specific slurring of speech which is readily separated from the forms of dysarthria already discussed.

Cortical dysarthria. An apractic hesitancy in word production, associated with difficulties in abstract, volitional movements of the lips and tongue is commonly associated with aphasia due to left frontal and temporal lesions. It never occurs as an isolated abnormality and the term should therefore not be used, since it leads to confusion.

Aphasia

If the patient's defect consists not so much in a disturbance of articulation as in an inability to use language, whether speaking, writing or comprehending it, then he is suffering from *aphasia*.

Aphasia may be classified or described in a number of different ways. from the clinical viewpoint it is necessary to ascertain, first, where the responsible lesion is likely to be and, second, what is the significance of the functional defect in language in relation to the patient's disabilities: how does it affect him?

Everyday use of language includes:

1. The ability to *use words* in spoken speech. This includes *articulation*, *fluency* (the ability to put words together into phrases and sentences of varying complexity, in various grammatical constructions, without hesitations and errors), *naming* and ready accurate *repetition* of complex statements and concepts.
2. The ability to *comprehend* spoken speech.
3. The ability to *read* to oneself (not aloud).

4. The ability to *write*.

5. The ability to comprehend *other symbols*, e.g. mathematical or musical symbols.

Speech defects may readily be analysed in these terms as disturbances of articulation, fluency, verbal comprehension, naming, repetition, reading and writing. If a simple rough score is given to each of these categories (Fig. 45), a broad analysis of speech function which has some

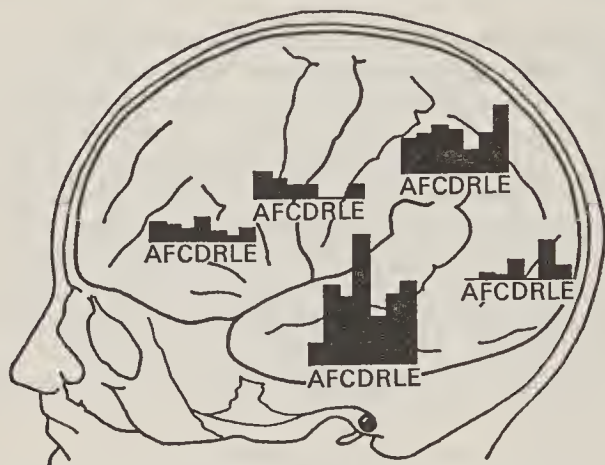


Fig. 45. The average degree of disturbance of various language modalities which occurs when there is an isolated lesion of various lobes (frontal, rolandic, parietal, temporal and occipital). A, articulatory disturbances; F, disturbances in the fluency of speech; C, disturbances of verbal comprehension; D, Disturbances of naming; R, disturbances of repetition; L, disturbances of reading; E, disturbances of writing. (After H. Hecaen & R. Angelergues (1964) in *Ciba Foundation Symposium on Disorders of Language*. London: Churchill)

localizing value can be built up. It can be seen for example, that disturbances of fluency, verbal comprehension, repetition and writing, are all prominent in left anterior temporal lobe lesions (amnesic or Wernicke's aphasia). Frontal lesions affect articulation and fluency more than the other categories of language. Parieto-occipital lesions impair reading (visual language functions) and parietal lesions impair several associative functions, but particularly writing.

Spoken speech

It will be noted that assessment of articulation and fluency, on the one hand, and reading and writing on the other, enables separation of lesions in front of and behind the central sulcus (Rolandic fissure): this

is a useful clinical concept which may be referred to as the 'anterior' and 'posterior' aphasic syndromes. Use of the terms 'receptive' and 'expressive' aphasia is to be discouraged because, not only does this classification not allow insight into the nature of the patient's functional defect (since it ignores the equally important functions of reading, writing, repetition, articulation and fluency), but it provides little information of localizing value. Similarly, an excessive concern with 'pure' varieties of aphasia and the establishment of theoretical centres for various aspects of language leads to great complexity and confusion, with little practical benefit.

A simple scheme for the practical examination of the aphasic patient follows:

First find out whether the patient's hearing is good. Roughly assess his speech disorder in the context of an ordinary conversation about everyday things. Note the presence of hesitations, the searching for a forgotten word, the compensation by the use of a descriptive phrase, replacing that which was forgotten (*paraphasia*), neologisms (invented or nonsense words), inappropriate words or phrases, syntactical and semantic errors. Note also any articulatory disturbances, particularly clumsiness and difficulty in coordinating movements of lips and tongue (orofacial apraxia). This is usually very pronounced when the patient is commanded to put out his tongue or close his eyes and these movements should not, therefore, be used to establish comprehension of speech or the patient's cooperation. Disturbances of fluency may also be noted in this way.

Formal tests of the ability to comprehend and carry out complex commands may then be useful. Inability to comprehend spoken words may be regarded as a special form of *auditory agnosia* (word deafness).

Spontaneous speech production. If the patient can use only a few words, make a note of what these are. If he repeats any word or phrase again and again (*perseveration* or *repetitive utterance*), note what it is.

If he has a considerable vocabulary, first make a note of any examples of lalling, slurring, etc., as an indication of *articulation*. Test him with such words and phrases as 'British Constitution', 'West Register Street', 'biblical criticism', 'artillery'. Then show him common objects—a knife, a pen, a matchbox, etc.—and ask him to *name* them or, if he is speechless, to indicate with his fingers the number of syllables in the name of each. Sometimes the patient has a general idea of the word he wants to use, but forgets exactly how to pronounce it; he omits some syllables or substitutes others for them, so that the listener may hardly be able to make out what word he wishes to use. These are signs of *amnesic* or *nominal* aphasia.

If he makes mistakes in his use of words, calling the knife a pen, he is

suffering from *word substitution*. In that case, one should note whether or not the patient shows that he is aware of his error by trying to correct himself, or whether he continues with similar errors or even uses nonsense words without realizing that they are not real words (neologisms). This is called *jargon aphasia* and indicates a lesion situated posteriorly in the temporal lobe.

Speech repetition. Ask him to repeat words after you. If necessary try to make clear your request by the aid of pantomime, repeating the word or phrase slowly and clearly several times. Remember never to shout at an aphasic patient: his hearing is normal. If he is able to repeat what you say, endeavour to find out whether or not he understands what he is saying.

Written speech

Speech comprehension. Ascertain whether or not his sight is good. If so, write on a piece of paper such questions or commands as: How old are you? Put out your tongue. If he does not respond satisfactorily, there is some word blindness present. Inability to read is called *alexia*.

Production. Ask him to write his name. (This can often be done when all other power of writing is lost.) If he is able to do so, ask him some simple question—How many do two and two make?—and get him to write a reply. If he has poor verbal comprehension put your question in writing. If his right hand is paralysed, make him write or print with his left. If he writes well, get him to write an account of his illness and note whether he makes use of the wrong word at times or whether there is repeated use of any particular word.

Can he write to dictation or copy words and sentences? Try, using a newspaper. If he succeeds, does he understand the meaning of what he writes?

Comprehension of other symbols. Write down certain numerals:

$$\begin{array}{r} 2 \\ +2 \\ \hline 4 \end{array}$$

$$\begin{array}{r} 2 \\ +2 \\ \hline 5 \end{array}$$

$$\begin{array}{r} 2 \\ +2 \\ \hline 6 \end{array}$$

and ask him to point out which is correct. Inability to understand and manipulate mathematical symbols, termed *acalculia*, may occur in posterior parietal lesions affecting the dominant hemisphere. If he can read music, test him with musical notes.

Gesture. Many aphasic patients make attempts to use gesture to com-

municate, but this too is usually defective. Furthermore these patients do not readily understand complex gestural instructions.

Use of common objects. Occasionally a patient who has neither motor nor sensory paralysis, nor ataxia, cannot perform certain acts, though he can easily execute their component movements. He is consequently unable to make use of objects, though he can recognize their use. This is known as *apraxia*. It results from damage to the parietal cortex or white matter of the left or of both hemispheres, or from disease of the connexions between the two hemispheres through the corpus callosum, and of the left parietal lobe. It affects only the left limbs, i.e. the right hemisphere, when the callosal fibres only are injured, but it is usually bilateral. It may be tested for by asking the patient to use certain objects, or make or imitate certain movements. For instance he may be given a box of matches and a cigarette and asked to light the latter. If there is apraxia, he may fail to open the box, or to take a match from it, or to strike the match, or even to light the cigarette with the match if he has succeeded in striking it. It is, of course, important to make sure that the patient understands the request.

THE CRANIAL NERVES

In this section a brief *résumé* of the essential points in the anatomy of each cranial nerve will be given indicating its function and, in some cases, the symptoms resulting from lesions affecting it. The method for clinical examination of each cranial nerve will be described.

First or olfactory nerve

Anatomy. The central processes of the bipolar sensory cells in the olfactory epithelium pass through the cribriform plate to the olfactory bulb, where the cells of the second olfactory neurones lie. Nerve fibres pass thence to the olfactory area of the cerebral cortex, the uncus of the parahippocampal gyrus.

Test

Have three small bottles containing some oil of cloves, some oil of peppermint and some tincture of asafoetida. Apply these to each nostril separately and ask the patient if he recognizes them. In testing, avoid the use of such irritating substances as ammonia, for these act partially through the 5th nerve. The sense of smell may be abolished. This is known as *anosmia*. Before concluding that this is due to neurological disease exclude local changes in the nose itself, e.g. catarrh. *Parosmia* is the name applied to that condition in which the sense of smell is perverted, so that, for instance, offensive substances seem to have a pleasant odour and vice versa.

Inquire also regarding *hallucinations of smell*. These sometimes constitute the aura of an epileptic fit.

Second or optic nerve

Anatomy. From the retina, the fibres of the optic nerve pass back to the optic chiasma. Here the fibres from the inner half of each retina decussate, whilst those from the outer half remain on the same side. Each optic tract, therefore, consists of fibres from the outer half of the retina on the same side and the inner half of the retina on the opposite side. Each tract passes back to the superior colliculus, the lateral geniculate body and the pulvinar of the thalamus of the same side. In these most of the fibres of the optic tracts terminate. A further system of fibres, which is known as the optic radiation, takes origin in the lateral geniculate body and passes through the posterior limb of the internal capsule and then backwards to the cortex around the calcarine sulcus. Those subserving the upper visual fields pass down through the substance of the temporal lobe, whilst those mediating impulses from the lower field pass up into the parietal lobe. The occipital cortex around the calcarine fissure constitutes the chief visual centre, and represents the opposite half of the field of vision, the left half of the field of vision being represented in the cortex of the right hemisphere and vice versa (Fig. 46).

Test

In testing the optic nerve, one has to investigate three functions: (*a*) visual acuity; (*b*) visual fields; and (*c*) colour sense.

Certain preliminaries must always be conducted. One of these is to see that any error of refraction in the patient's eye is first corrected and that also there is no gross disease of the structure of the eye; another is to take care to examine each eye separately.

Visual acuity. For technique of testing, see p. 267.

Visual fields. When we fix the eye upon an object, we not only see that object, but also a number of objects in the neighbourhood more or less distinctly. The full extent of this vision is measured in assessment of the visual fields. It should be noted, however, that the field of vision is limited both by the area of sensitive retina and by the margins of the orbit, nose and cheek. Hence the position of the eye is important. A rough estimate of the extent of the fields of vision for large objects may be obtained in the following way.

Seat yourself opposite to the patient and at a distance of about half a metre from him. If his right eye is to be tested, ask him to place his hand of his left and look steadily at your own *left* eye. Look steadily yourself at the patient's right eye, your own right being closed, and hold up your left hand in a plane midway between his face and your own, and at first

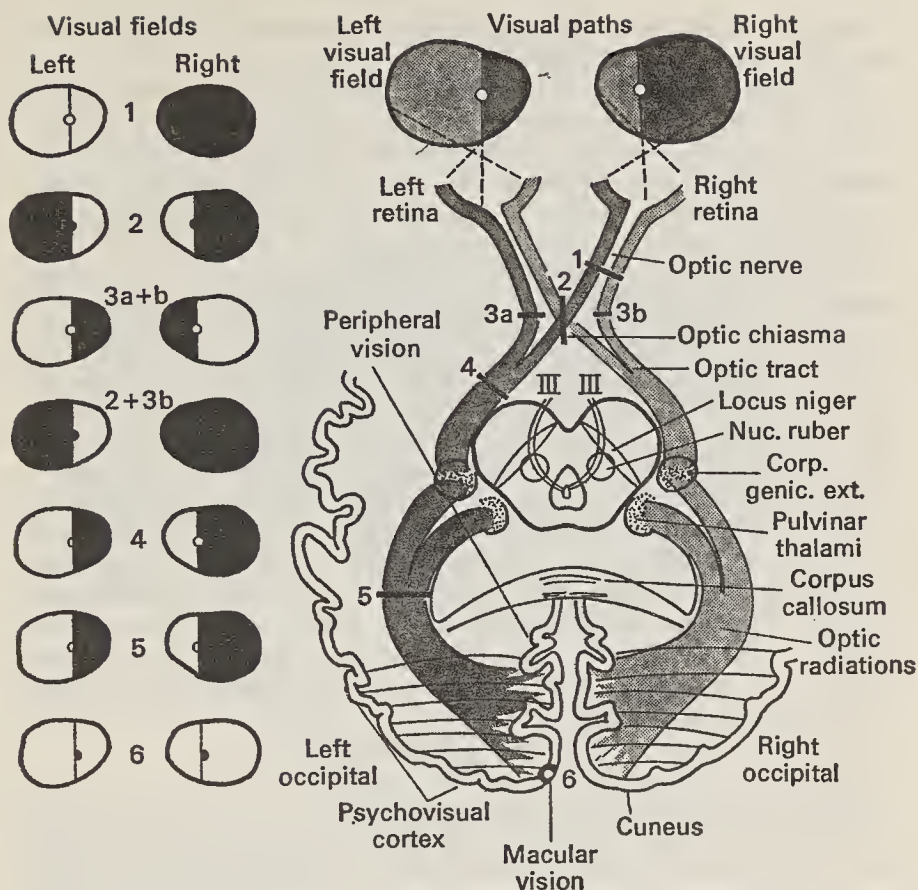


Fig. 46. The visual pathways. Lesion at 1 produces complete blindness in the right eye with loss of direct right reflex. Lesion at 2 produces bitemporal hemianopia. Lesions at 3a and 3b produce binasal hemianopia. Lesions at 2 and 3b produce blindness of the right eye with temporal hemianopia of the left visual field. Lesion at 4 produces right homonymous hemianopia, with hemianopic pupillary response. Lesion at 5 produces right homonymous hemianopia, with normal pupillary reaction to light. Lesion at 6 produces right homonymous central hemiscotoma.

at almost full arm's length to the side. Keep moving the fingers of the hand and bring it nearer until you yourself can just 'with the tail of your eye' catch the movements of the fingers. Then ask the patient whether he sees them, telling him meanwhile to be sure not to take his own eye off yours. If he fails to see the fingers, keep bringing the hand nearer until he does see them. Test the field in this fashion in every direction—

upwards, downwards to right, and to left—using the extent of your own field for the purpose of comparison.

This gives the outline of his field for appreciation of a moving object, which may, however, be relatively intact when the fields for other forms of stimulation are seriously constricted. In consequence, his field for appreciation of a stationary object must also be investigated in a similar manner, but by asking the patient to indicate when he sees the observer's fingers held at rest.

Considering the field of vision in more detail, we appreciate that whereas the objects which cause images to fall upon the central part of the retina (the macula) are seen in minute detail and bright colouring, objects farther and farther from the point of fixation are seen with less and less distinction and colour, until at the periphery of the field we can only appreciate the presence of an object of considerable size without being able to judge its form. To the temporal side of the central point of vision is the *blind spot* which represents the optic nerve head area in which there are no light receptors. Perimetry surveys the field of vision and the limits of perception are charted. The centre point of the chart corresponds to the visual axis; the point of fixation is therefore the point of more distinct vision. Around this point are arranged a series of more or less concentric lines, each of which denotes equal visual acuity, and is called an *isopter*. For purposes of investigation we divide the visual field into three parts:

1. An area surrounding the point of fixation to 20° .
2. An intermediate area between 20° and 50° .
3. An outer area from 50° to the periphery.

The fixation point is not exactly central so that the outer and inner part of the field is unequally divided. Further, the boundary is delimited inwards and upwards by the nose and brow. Testing with a 5 mm object we find the extent of the average field of vision is 100° outwards, 60° upwards and inwards and 75° downwards. The field charted with a 20 mm object is shown in Fig. 47; note the restriction of the lower nasal field by the bridge of the nose.

The binocular field extends 200° or more laterally and about 140° vertically, in the middle of which is a circular portion common to each eye with a diameter of about 120° . On each side of this paired area is a semilunar area which is unpaired and which accounts for the remainder of the field.

Perimetry is concerned with an investigation of the uniocular field of vision. We have noted that the visual acuity is very much lower at the periphery and gradually increases as we pass to the point of fixation. We may test this acuity with objects of different size, and we shall find that whereas a very small object is visible at and near the fixation point, it fades from view as it is withdrawn towards the periphery. By using a

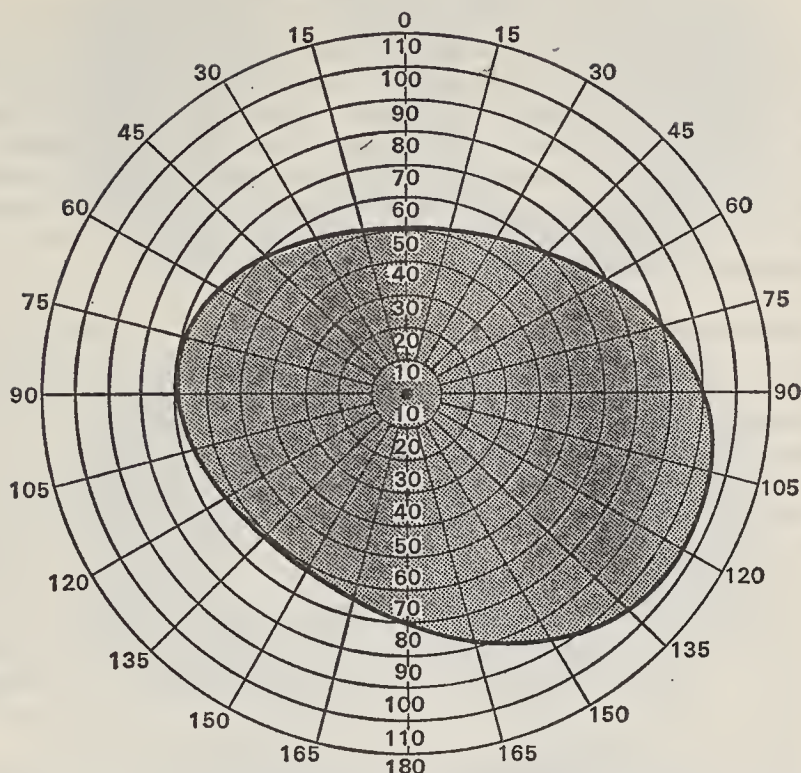


Fig. 47. The extent of the right field of vision with a white target of 20 mm diameter mapped on a perimeter at a distance of 330 mm.

graduated series of objects, we are able to plot out a series of isopters each of which corresponds to a known size of object used at a known distance from the eye.

That part of the field between the periphery and the 30° circle is investigated by means of a perimeter. A 3 mm white test object is usually used, but when visual acuity is impaired, a larger diameter may be required. Thus the fraction of diameter of object/distance (usually $3/330$ mm) is a measure of the visual acuity at the particular point on the field which is being tested.

The area within the 30° circle is examined by means of test objects upon a black screen—*Bjerrum's screen*—at a distance from the patient of 1000 or 2000 mm.

The patient is seated comfortably at this distance with the head steadied by a chin and head rest, and a grey object 1 cm in diameter with a black centre is fixed to the screen on a level with the patient's eye.

The blind spot is first of all mapped out with a white object 20–30 mm in diameter. The peripheral field is next mapped out with a 10 mm object, and at a distance of 2000 mm it should be circular and extend to about 25° , that is to the edge of the 2 m² screen. With the small object areas of blindness or defective perception should be sought around the blind spot, especially between this area and the macula, the centrocaecal area, and in the horizontal meridian on the nasal side of the fixation spot. The findings are marked upon the screen with black pins, and subsequently transferred to a chart.

Changes in the field of vision. It may be contracted all round its periphery. This is spoken of as *concentric diminution* of the field of vision. It occurs in long-standing papilloedema, some forms of optic atrophy, bilateral lesions of the anterior part of the cortical visual centres, various affections of the retina and hysteria.

Sometimes the loss of vision is confined to the centre of the field. This is spoken of as a *central scotoma*. Sometimes it is due to local disease of the choroid or of the retina in the neighbourhood of the macula. In that case it may affect only one eye. A unilateral central scotoma is also produced by retrobulbar neuritis, which in most cases is a symptom of multiple sclerosis. More rarely, it may be due to toxic causes or vitamin deficiency, when it is generally bilateral. Pressure on the optic nerve is another cause. It may also result from a lesion of the posterior part of the cortical visual centres and is then bilateral, but this is rare.

The term *hemianopia* means loss of sight in one-half of a visual field. When the same half of both fields of vision is lost, the hemianopia is described as homonymous, e.g. right homonymous hemianopia when the blindness occupies the right half of both the right and left field (Fig. 47).

Superior and inferior hemianopia mean loss of the upper and lower halves of the visual field respectively. They are of rarer occurrence than the lateral variety and are sometimes spoken of as altitudinal hemianopia. Hemianopia limited to one quadrant of the field is described as quadrantic hemianopia or quadrantanopia.

Bitemporal hemianopia means loss of vision in the temporal or outer halves of both fields, and is due, therefore, to loss of function of the nasal half of each retina. It can only be produced by a lesion of the optic chiasma, involving those fibres of the optic nerves which decussate, and is accordingly rare. It is commonly due to a tumour of the pituitary body but may be produced by inflammatory or traumatic lesions of the optic chiasma (Fig. 48).

Binasal hemianopia signifies a loss of the nasal or inner half of each field and indicates a diminution of visual function of the temporal half of each retina. It can be produced by a bilateral lesion confined to the

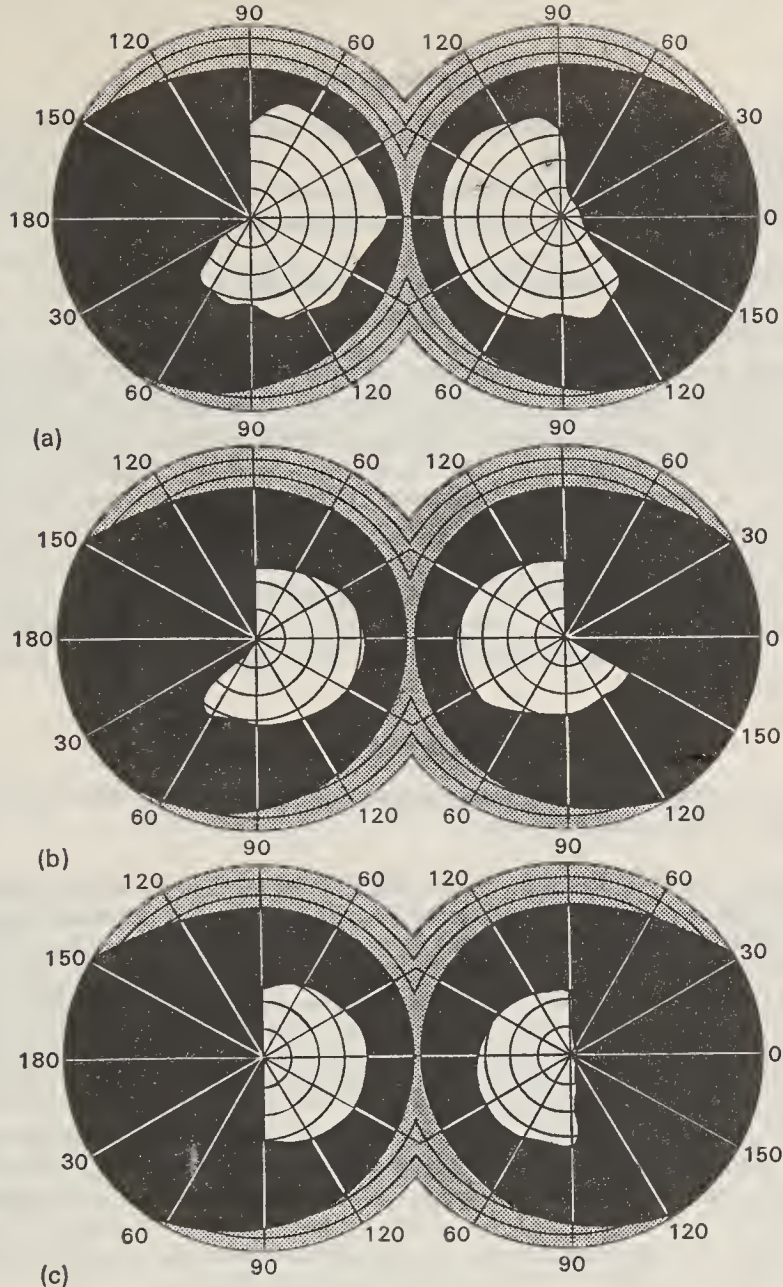


Fig. 48. Fields of vision in a case of pituitary tumour (probably chromophobe adenoma) showing the development of bitemporal hemianopia. (a) May 1947. VA Lt 6/12 Rt 6/18. (b) August 1947. VA Lt 6/12 Rt 6/12. (c) October 1947. VA Lt 6/12 Rt 6/12. The fields were plotted on a Bjerrum's screen at a distance of 20 m from the eye to the point of fixation at the centre of the screen, using a 10 mm white object. The black areas show the average normal field of vision and the white areas the patient's fields. (By courtesy of D. W. C. Northfield)

uncrossed optic fibres on each side of the chiasma, and may also occur in open-angle glaucoma.

Temporal and nasal hemianopia are sometimes described as heteronymous, in contradistinction to the homonymous variety.

Colour sense. For method of testing see p. 268.

Subjective visual sensations

Among the commonest subjective visual sensations is the occurrence of what are known as *muscae volitantes*—little specks seen floating before the eyes, especially on looking at a white surface or up to the sky. They commonly occur in normal subjects and are frequently complaints in anaemic and debilitated persons. In migraine, peculiar zigzag lines, known as ‘fortification figures’ or *teichopsia* are often seen at the beginning of the attack and in the investigation of such a case they should always be inquired for. Visual hallucinations occur in a number of neurological diseases, notably in delirium tremens and in temporal and occipital lobe disorder; they may also form part of the aura in epilepsy.

Third, fourth and sixth nerves

It is convenient to take these together, as together they innervate the muscles which move the eye.

Anatomy. The fibres of these nerves take their origin from a series of nuclei which begin in the floor of the cerebral aqueduct below the superior corpora quadrigemina, and extend down as far as the eminentia teres in the floor of the 4th ventricle. The nucleus for the 3rd nerve is farthest forward; its most rostral nerve cells supply the ciliary muscle and iris (Edinger-Westphal nucleus), those for the ocular muscles being more caudally situated. Caudal to this is the nucleus of the 4th, and, most caudal of all, that of the 6th. The 3rd nerve emerges on the inner aspect of the crus, and is therefore likely to be involved in lesions of that part of the brain.

The trochlear nerves emerge on the anterior part of the roof of the 4th ventricle. They are peculiar in that they are the only cranial nerves which decussate between their nuclei and their point of emergence, and that they emerge dorsally.

The 6th nerve emerges between the medulla and pons. Its long intracranial course renders it particularly liable to the effects of pressure.

Functions

The 6th nerve supplies the external rectus, the 4th supplies the superior oblique. All the other extraocular muscles, the sphincter pupillae, the muscle of accommodation, and the levator palpebrae superioris, are supplied by the 3rd.

Ocular movements

Horizontal movement outwards is described as abduction, inwards adduction; vertical movement upwards as elevation and downwards as depression. The eye is also capable of diagonal movements at any intermediate angle. Rotatory movements, the eye rolling like a wheel towards the nose (internal rotation) or away from the nose (external rotation), do not occur normally but may be seen in some varieties of ocular palsy. Note that the *recti act as elevators and depressors alone when the eye is in abduction, and the obliques act similarly when the eye is in adduction* (Fig. 49). Their function may therefore be easily assessed

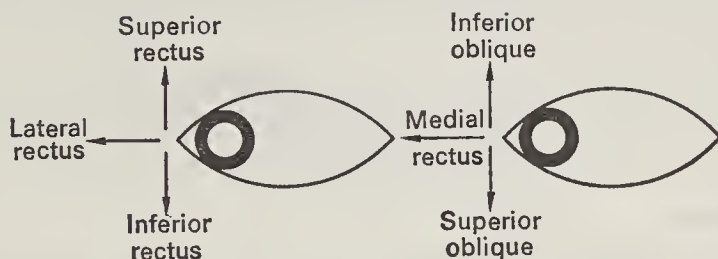


Fig. 49. The action of the external ocular muscles.

by testing the movements of elevation and of depression in both full abduction and full adduction. This is more important than simply testing elevation and depression in the mid-position of gaze. The internal and external recti always act directly in a single plane, but all movements require the coordinated activity of the whole group of extra-ocular muscles.

Normally the movements of the two eyes are symmetrical, so that the visual axes meet at the point at which the eyes are looking. This is spoken of as conjugate movement of the eyes. *Infranuclear* (lower motor neurone) lesions of the 3rd, 4th and 6th nerves lead to paralysis of individual eye muscles or groups of muscles. *Supranuclear* (upper motor neurone) lesions lead to paralysis of conjugate movements of the eyes.

Lower motor neurone (infranuclear) lesions

Sixth nerve. Inability to move the eye outwards and diplopia on looking in that direction. Possibly internal squint.

Fourth nerve. Impaired power of downward movement, and on attempting to look downwards in the mid-position of gaze the eyeball is

rotated inwards by the unopposed action of the inferior rectus (Fig. 49). Diplopia only below the horizontal plane, with the images uncrossed, but the false one tilted. There is rarely a visible squint.

Third nerve. Ptosis. The eye is displaced downwards and outwards and further movement is only possible outwards and a little downwards. Pupil usually dilated and fixed. Loss of power of accommodation.

Paralysis of the 3rd nerve is not infrequently partial—only one or a few of these functions being lost.

It must be remembered that smooth muscle in the upper lid is innervated by the cervical sympathetic and exerts a tonic elevating action. Slight ptosis therefore occurs after a lesion of the cervical sympathetic but the pupil is then small, rather than large (Horner's syndrome) (p. 227).

Thus the signs of a lesion involving one or more of these nerves may be: (a) defective movement of the eye; (b) the presence of a squint; (c) the presence of diplopia; and (d) pupillary abnormalities. Of these signs diplopia and pupillary abnormalities are the most reliable.

Strabismus

By *squint* or *strabismus* is meant a condition in which the visual axes do not meet at the point of fixation. Of this there are two varieties, *paralytic* and *concomitant*, and it is necessary that the two be carefully distinguished.

Paralytic strabismus. The following are the characters of a paralytic squint:

1. *Limitation of movement.* Paralytic strabismus is due to weakness in one or more of the extra-ocular muscles; a prominent feature, therefore, is lack of ability to move the eye in the direction of the physiological action of the muscle affected. Although this weakness is usually apparent, it is sometimes so slight, or the unaffected muscles mask it so much, that the defective movement of the eye is hardly visible.

Movements in the so-called *cardinal directions* are tested by fixing the patient's chin with one hand and moving the forefinger of the other in the direction indicated. The eyes move normally 50° outwards, 50° inwards, 33° upwards and 50° downwards.

If an eye fails to move at all, or fails to move throughout the normal angular excursion, the deviation of the eye in a direction opposite to the physiological action of the muscle is called the *primary deviation* or *squint*, and it is measured by the angle which a line from the object to the nodal point of the eye makes with the visual axis. If now we cover the unaffected eye and so cause the patient to take on fixation with the affected eye, we shall find that the eye that is covered will deviate still

more than the primary deviation of the affected eye. This deviation of the healthy eye is the so-called *secondary deviation* or *squint*, and this difference in amount between the primary and secondary deviation is the most important distinguishing feature between paralytic and comitant strabismus.

2. *False orientation of the field of vision.* This is an erroneous judgement by the patient of the position of an object in that portion of the field of vision towards which the paralysed muscle should normally move the eye. Take the case of a patient who has paralysis of the right external rectus muscle. If such a patient closes the left eye and is asked to touch suddenly an object held in the horizontal direction on his right side, he may fail and strike wide of the object on his right-hand side.

3. *Dizziness* is occasionally a symptom of paralytic strabismus when both eyes are opened. It is due partly to the confusion of double sight and partly to false orientation.

4. *Double vision (diplopia).* In order to overcome double vision, the patient turns his head in the direction of the action of the paralysed muscle. Information may therefore be obtained as to which muscle is involved by noting the way in which the head is held.

Patients with paralytic squint complain of double vision, which is due to the fact that owing to the lack of movement of one eye in a particular direction the images of external objects do not fall upon 'identical points' of the two retinæ; this double vision or *diplopia* is therefore in that part of the field of vision into which the paralysed muscle should move the eye, were it unaffected.

In health, when fixing an object, the image formed in each eye falls upon the macula, so that not only are the two images of equal intensity and definition, but since they fall upon identical points of the two retinæ they produce only a single image. In paralytic strabismus, the image of the object fixed falls upon the macula of the healthy eye and is seen with distinctness and detail, and is called the *true image*, whereas in the affected eye the image falls upon the retina outside the macula, and as in consequence it is indistinct and blurred, it is called the *false image*. Most patients can therefore clearly recognize which of the two images they perceive is the true image and which the false.

The investigation of a case of paralytic strabismus and the diagnostic value of diplopia. First make certain that the diplopia is *binocular*, since certain conditions, for example, lens opacities and astigmatism, may produce *monocular diplopia*.

Movements should always be assessed as indicated in Fig. 49. These are the *cardinal* directions of gaze. In this way diplopia can be resolved into horizontal and vertical planes only, greatly simplifying its analysis and interpretation.

When the images in diplopia are separated laterally, so that the right

image belongs to the right eye and the left to the left eye, the condition is spoken of as *homonymous* or *uncrossed diplopia*. If, however, the left image belongs to the right eye and the right to the left, it is called *heteronymous* or *crossed diplopia*. It will also be found that the real image belongs to the healthy eye, whereas the false image belongs to the paralysed eye.

The production of homonymous diplopia. If, as the result of paralysis of an abductor muscle (external rectus muscle), there is deviation of the eye inwards (convergent strabismus), the image in this eye will fall upon a point of the retina internal to the macula. Two things will result: (a) the image will not be so sharp as the image in the healthy eye, proving that the *false* image belongs to the affected eye; and (b) since images that fall upon the retina on the nasal side of the macula are projected in space to the temporal side of the eye, it follows that paralysis of an abductor producing convergent strabismus causes homonymous diplopia and also, that the *false image is always projected in the direction of the physiological action of the paralysed muscle*.

The production of heteronymous or crossed diplopia. If, as the result of paralysis of an adductor muscle (internal rectus), there is a deviation of the eye outwards (divergent strabismus), the image in this eye will fall upon a point of the retina external to the macula. The false image is produced in the affected eye, and since images that fall upon the retina to the temporal side of the macula are projected into space to the nasal side of the eye, it follows that *paralysis of an adductor producing divergent strabismus causes heteronymous or crossed diplopia*.

In a similar way it may be shown that in paralysis of an elevator muscle the false image (which belongs to the affected eye) lies on a higher level than the true image, and in paralysis of a depressor muscle the false image lies below the true image.

If a rotator muscle is weak, the false image is tilted.

Method of finding the direction of maximum diplopia. The patient is seated with the head fixed in position (preferably in a head rest) with a red glass before the right eye, and a green glass before the left. At a distance of about 5 metres the observer moves a light in the direction indicated in Table 7, each of the lateral squares corresponding to a pair of true associated muscles; thus a maximum vertical diplopia produced when the patient looks up and to the right into the right superior square shows that either the right superior rectus or the left inferior oblique muscle is the one affected. It is only necessary to find to which eye the false image belongs to decide which muscle is paralysed. The higher of the two images indicates the affected eye.

Note that in using this chart the area of greatest vertical diplopia is the namesake of the affected muscle or its true associate in the other eye; further, that the true associates always bear names which are the most

TABLE 7. CHARTING THE FIELD OF DIPLOPIA IN A CASE OF PARALYTIC STRABISMUS.

<i>The patient looks</i>		
Upwards to the left <i>Left superior area</i>	Upwards <i>Superior median area</i>	Upwards to the right <i>Right superior area</i>
To the left <i>Left external area</i>	Straight ahead <i>Primary area</i>	To the right <i>Right external area</i>
Downwards to the right <i>Left inferior area</i>	Downwards <i>Inferior median area</i>	Downwards to the left <i>Right inferior area</i>

contrary possible, thus *left inferior oblique* is in every term opposite to *right superior rectus*.

Concomitant strabismus. It has been explained when dealing with paralytic squint that the amount of angular deviation of the two visual axes varies with different positions of the two eyes, and also that secondary deviation is always greater than primary deviation.

In concomitant strabismus, as its name implies, the angular deviation of the visual axes is the same in whatever position the eyes may be; in other words, the primary and secondary deviation are always equal.

The *cover test* is performed as follows. Ask the patient to look at an object immediately in front of him. Suddenly cover the apparently fixing eye and ask the patient to fix the object with the uncovered eye. If this eye makes any movement in taking up fixation, it must have been previously deviating. If now the eye behind the screen (which was previously fixing) is observed it will be seen to deviate in the same relative direction as was the other eye, and *to the same angular amount*, that is, the primary and secondary deviation are equal.

The *clinical features* of concomitant strabismus are:

1. It always begins in early childhood, over 70% before the fifth year and the great majority before 3 years of age.
2. The movements of the eyes are good in all directions.
3. Diplopia is practically never a symptom.
4. The primary and secondary deviation are equal.
5. The deviating eye usually has defective vision.

A squint may be *intermittent* or *constant*, and if constant, *monocular* when the same eye deviates whilst the other usually fixes, or *alternating* when either eye fixes indifferently.

Upper motor neurone (supranuclear) lesions

In addition to the defects of movement due to paralysis of the individual ocular muscles, weakness of paralysis of the movement of both eyes in one direction sometimes occurs. Thus the patient may be unable to look to either side, or upwards or downwards; or the power of convergence alone may be lost. Weakness of conjugate lateral movement may occur in hemiplegia due to cerebral lesions, especially in the acute stage. Palsy of this movement occurs with lesions in the neighbourhood of the 6th nucleus or the vestibular nucleus in the pons; the lesion is on the side to which the movement is weak. Bilateral paralysis of lateral conjugate movement may occur in centrally placed pontine lesions above the level of the 6th nerve nuclei. Palsies of conjugate upward gaze are always associated with disease of the central parts of the brain stem or inferior thalamic region, or disease in the neighbourhood of the oculomotor nuclei. Impaired downward gaze is very rare.

If both eyes are kept persistently turned in one direction, the condition is spoken of as tonic or maintained conjugate deviation of the eyes. It is usually either to the right or to the left. It may be due either to a lesion which produces paralysis or to one which causes irritation or spasm. In the former the eyes (and usually also the head) are turned towards the side of the lesion, provided the lesion is in the cerebral hemisphere. The patient 'looks towards his lesion'. An irritative lesion in a similar situation causes deviation towards the healthy side. If, however, the lesion is in the pons, these rules are reversed, the deviation being towards the sound side in a paralytic lesion and towards the affected side in one which is irritative. Irritative lesions in the brain stem, it should be remembered, are very rare.

Skew deviation of the eyes—in which, for example, one is directed upwards and the other downwards—occurs in lesions of the labyrinth and in cerebellar disease. Other abnormal ocular movements, such as *ocular bobbing*, *opsoclonus* and *ocular dysmetria* are rare. Abnormal *saccadic* (cogwheel) jerky movements of the eye during voluntary conjugate deviation occur in certain diseases of the basal ganglia; they are especially characteristic of *Parkinson's disease*.

Internuclear ophthalmoplegia is due to a lesion which destroys one medial longitudinal fasciculus in the midbrain or upper pons. On attempted lateral gaze there is pronounced rhythmic nystagmus of the abducting eye and impaired medial deviation of the adducting eye. All other gaze movements and the pupils are normal. The lesion is on the side of the impaired adduction, not on the side of the nystagmus. This sign is important because it is common in multiple sclerosis.

The term *nystagmus* is applied to a disturbance of ocular movement characterized by involuntary, often rhythmical oscillations of the eyes.

These movements may be horizontal, vertical or rotary. The speed of the movements may be the same in both directions, or quicker in one direction than another; in the latter case the quicker movement indicates the direction of the nystagmus. To examine for nystagmus, ask the patient to look straight in front of him and observe whether the eyes remain steady. Then ask him to look or to follow your finger to his extreme right, then to the left, and then upwards and downwards. Observe the rate, amplitude and rhythm of the movements in each direction. Some forms of nystagmus, particularly that associated with benign epidemic vertigo, and with posterior fossa neoplasms, may be induced only by certain movements of the head (positional nystagmus).

A few irregular jerks of the eyes are often seen in full lateral deviation in normal subjects. The brief duration and irregularity of these movements distinguish them from true nystagmus.

Nystagmus is most commonly due to disorders of the vestibular system (either centrally or peripherally), lesions affecting the central pathways concerned in ocular movements, e.g. the medial longitudinal fasciculus, or to weakness of the ocular muscles. Nystagmus of visual origin is pendular and often rotary on central fixation of the eyes. Congenital nystagmus also shows this pendular quality.

Examination of the pupils

The following points must be noted about the pupils in every case:

Size. Compare the size of the two pupils, first in a bright light and then in a dim light. Note whether the pupils are large or small and whether any irregularity is present. The size of the pupil in health is very variable. As a rule, the pupils are larger in dark eyes than in light and they tend to be small in elderly subjects. Slight inequality of the pupils may also be present in perfectly healthy subjects.

If one pupil is larger than the other, one must decide which is the normal. This is not always very easily answered, but the pupil which is less mobile is usually the abnormal one.

Shape. Note whether the pupil is circular in outline, as it should be, or whether its contour is irregular. Such irregularities may be due to adhesion of the iris to the lens, as a result of an old iritis, or to neurosyphilis.

Mobility. The *reaction to light* is a reflex. The afferent fibres involved are contained in the optic nerve, travelling to the oculomotor nuclei whence the efferent fibres pass by the 3rd nerve, through the ciliary ganglion, to the pupillary sphincter.

Examine each eye separately. Place the patient opposite a bright light, be sure his accommodation is relaxed and cover the eye with the hand. Leave it covered until the pupil dilates, then withdraw the hand and watch the pupil closely. It should contract almost immediately, then dilate again a little, and, after undergoing slight oscillations, settle down to its normal size. The test may also be carried out by shining a bright light into one eye and then the other.

Owing to the decussation of some of the fibres in the optic nerves at the optic chiasm, light shone into one eye stimulates the brain stem nuclei (3rd nerve) concerned with pupillary constriction bilaterally. This pathway is pregeniculate. As a consequence, if light is shut off from one eye both pupils dilate, and if bright light is made to enter one eye both pupils contract. This is known as the *consensual reaction* of the pupils. It should be tested by keeping one eye in the shade while the other is tested. The effect on the pupil of the shaded eye is then observed.

Reaction to accommodation. The pupils become smaller on accommodating for a near object (*miosis*). Convergence of the eyes, accommodation and miosis are due to closely related reflexes.

Hold up one finger close to the patient's nose. Ask him to look away at a distant object. Then ask him to look quickly at your finger. As the eyes converge to accomplish this the pupils become smaller. If the patient is blind, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face, and then asking him to 'look at the finger'.

The *Argyll Robertson pupil* is the classical pupillary abnormality in neurosyphilis. The term is used to describe a pupil which is small and irregular, and which reacts briskly to accommodation but which does not react to light directly or consensually. The pupil dilates slowly or imperfectly to atropine. The abnormality is typically bilateral but it is frequently more obvious on one side.

Hippus is a term applied to alternate rhythmic dilatation and constriction of the pupil, either in response to light or spontaneously. It is rarely of clinical significance, although it may be unusually prominent in cases of retrobulbar neuritis.

Adie's pupil is an abnormality characterized by absent or delayed pupillary constriction with light or with accommodation/convergence. Once constricted the pupil dilates only very slowly, either in response to darkness or to far gaze. The abnormality is thus variable and this has led to its confusion with the Argyll Robertson syndrome. The pupil in Adie's syndrome varies in size from day to day but *never* reacts promptly to light. It is frequently unilateral and sometimes associated with absent tendon reflexes, often on the same side (Holmes-Adie syndrome). The distinction between the Argyll Robertson and Adie pupillary abnormalities is important since the latter, probably due to parasympathetic denervation is usually of no clinical significance. The distinction may be made by instillation of sterile 2% methacholine into the conjunctival

sac: the Adie pupil constricts but the Argyll Robertson pupil does not.

Paralysis of the cervical sympathetic (Horner's syndrome). The sympathetic nerve fibres supplying the pupil take origin in the lower cervical and upper thoracic regions of the spinal cord (ciliospinal centre), from which they emerge in the first thoracic nerve roots and pass to the sympathetic cord by the rami communicantes. From the cervical sympathetic chain the fibres pass along the internal carotid to the cavernous plexus, and thence via the ophthalmic division of the 5th to the eyeball. They convey the impulses which cause dilatation of the pupil and also supply the unstriated muscle in the insertion of the levator palpebrae into the upper lid.

Paralysis of the cervical sympathetic is recognized by the following signs: apparent enophthalmos; slight drooping of the upper lid, due to paralysis of the unstriated muscle fibres contained in it; contraction of the pupil with absence of dilatation on shading the eye or on instillation of cocaine; abolition of the ciliospinal reflex; and, less commonly, absence of sweating, even after the use of pilocarpine, on the corresponding half of the head and neck, both in front and behind, extending as low as the 3rd rib and 3rd thoracic spine, and over the whole of the upper limb on the same side.

Dilatation of the normal pupil when the skin of the neck is pinched is due to reflex excitation of the pupil dilating fibres in the cervical sympathetic, and is abolished by lesions of that nerve and by some medullary cervical and upper thoracic cord lesions.

Fifth or trigeminal nerve

Anatomy. The *sensory root* takes origin from nerve cells in the Gasserian ganglion and enters the lateral surface of the pons at about its middle. The fibres which conduct impulses for light touch and postural sensibility terminate in a large nucleus in the pons situated lateral to the motor nucleus near the floor of the 4th ventricle, while the fibres for pain and thermal sensibility terminate in the 'descending' or bulbospinal root, which extends as low down as the 2nd cervical segment of the cord. Immediately distal to the trigeminal ganglion the nerve separates into its three divisions.

The *first or ophthalmic division* supplies the conjunctiva (except that of the lower lid), the lacrimal gland, the mesial part of the skin of the nose as far as its tip, the upper eyelids, the forehead, and the scalp as far as the vertex.

Lesions of this division result in loss of cutaneous and corneal sensibility and may cause trophic changes in the cornea, *neuropathic keratitis*. The corneal reflex is abolished.

The *second or maxillary division* supplies the cheek, the front of the temple, the lower eyelid and its conjunctiva, the side of the nose, the upper lip, the upper teeth, the mucous membrane of the nose, the upper part of the pharynx, the roof of the mouth, most of the soft palate, and the tonsils.

Lesions of this division lead to loss of sensation in the above area and sometimes to loss of the palatal reflex.

The *third or mandibular division* is joined by the motor root. It supplies sensation to the lower part of the face, the lower lip, the ear, the tongue, and the lower teeth. It also supplies parasympathetic fibres to the salivary glands. The motor division supplies the muscles of mastication.

The *motor root* takes origin in a small nucleus lying medial to the chief sensory nucleus, and partly also from the mesencephalic root, which arises in the nerve cells scattered around the cerebral aqueduct. It emerges at the side of the pons, just anterior to the sensory division, passes underneath the trigeminal ganglion, and joins the mandibular division.

Paralysis of the whole 5th nerve leads to loss of sensation in the skin and mucous membrane of the face and nasopharynx as described above (Fig. 50). The salivary, buccal and lacrimal secretions may be much diminished and trophic lesions may be present. Taste is preserved, but lack of secretions may result in its subjective impairment.

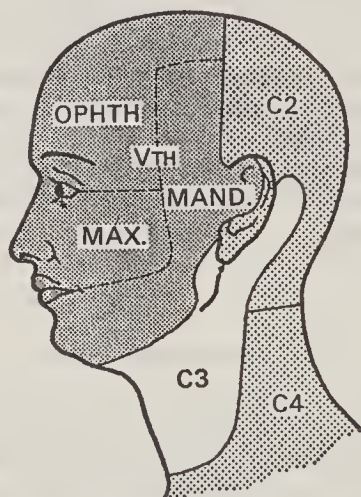


Fig. 50. Lateral view of the skin areas supplied by the Vth cranial nerve and the 2nd, 3rd and 4th cervical segments.

Testing the motor functions

Ask the patient to clench his teeth: the temporal and masseter muscles should stand out with equal prominence on each side. This is a sign better checked by palpation than by inspection. If there is paralysis on one side, the muscles on that side will fail to become prominent and, on opening the mouth, the jaw will deviate towards the paralysed side being pushed over by the healthy lateral pterygoid muscles.

Testing the sensory functions

The sensibility of the area supplied is tested in the usual way (p. 225).

Taste. In suspected lesions of the 5th nerve the sense of taste should always be examined. These fibres pass from the lingual nerve into the chorda tympani and thence through the geniculate ganglion of the facial nerve and the nervus intermedius into the medulla oblongata. Rarely, these taste fibres may enter the brain stem with fibres in the maxillary division of the trigeminal nerve, rather than through the chorda tympani and the facial nerve. The taste fibres from the posterior third of the tongue enter by the glossopharyngeal nerve.

All the taste fibres enter the tractus solitarius and relay in the nucleus of the tract, whence further fibres pass upward in the gustatory fillets to the thalamus and thence to the foot of the post central gyrus. *Ageusia* or loss of taste occurs with lesions of the peripheral pathways concerned or with centrally placed pontine lesions which may involve the gustatory lemniscus.

To test the sense of taste, use strong solutions of sugar and common salt, and weak solutions of citric acid and quinine. These are applied by a glass rod to the surface of the protruded tongue and if the taste is recognized the patient writes down 'sweet', 'salt', 'sour' or 'bitter', as the case may be, *without* withdrawing the tongue. After each test the mouth must be rinsed. The quinine test should be applied last, as its effect is more permanent than that of the others. Both the anterior and posterior parts of the tongue should be tested. Taste may also be assessed using a weak galvanic current. Loss of taste may arise from lesions of the taste fibres in any part of their course. In addition to loss of taste, one should always ask the patient whether he has any abnormal taste sensations. These may form the aura of an epileptic fit, as in temporal lobe epilepsy.

Testing the corneal reflex

Twist a light wisp of clean cotton wool or cotton thread into a fine hair and lightly touch the lateral edge of the cornea at its conjunctival margin with the wisp, having asked the patient to gaze into the distance or at the ceiling. If the reflex is present the patient blinks. It is helpful to steady the hand by gently resting the little finger on the patient's cheek. The cornea should not be wiped with the cotton and the central part of the cornea should never be touched, since to do so in the presence of corneal anaesthesia carries the risk of corneal ulceration and subsequent scarring. The two sides should be compared. This can sometimes be done more easily by lightly blowing a puff of air into each cornea in turn.

Seventh or facial nerve

Anatomy. The course of the fibres from the cortex to the nucleus of this nerve has already been described (p. 192). The nucleus is situated in the pons lateral to that of the 6th nerve. On leaving the nucleus the fibres wind round the nucleus of the 6th, and emerge medial to the 8th nerve, between the olive and restiform bodies.

The nerve then lies in close contact with the 8th and enters the internal auditory meatus with it so that a lesion of the one at this part often also affects the other. During its course through the temporal lobe, in close proximity to the aditus of the tympanic antrum, it gives off a branch to the stapedius muscle. It is joined by the chorda tympani which contains taste fibres from the anterior two-thirds of the tongue at the geniculate ganglion. In this part of its course the nerve is vulnerable to trauma and to oedema, since it is enclosed in a bony tube. It emerges at a point opposite the junction of the anterior border of the mastoid with the ear, and spreads out on the side of the face to supply the facial muscles.

Functions. The 7th is almost entirely a motor nerve. It supplies all the muscles of the face and scalp, except the levator palpebrae superioris. It also supplies the platysma. The chorda tympani travels with the facial nerve during part of its course and taste may therefore be lost on the anterior two-thirds of the tongue with lesions of the proximal part of the nerve. There is sometimes a small area of altered cutaneous sensation at the auricle in such cases.

Effects of paralysis. These are usually at once seen on looking at the patient. The affected side of the face has lost its expression. The nasolabial fold is less pronounced, the furrows of the brow are smoothed out, the eye is *more* widely open than the other, and the mouth is somewhat drawn to the healthy side. The patient is unable to whistle; food is apt to collect between his teeth and his gums, and saliva and any fluid he drinks may escape from the affected angle of the mouth.

Testing the seventh nerve

1. Ask the patient to shut his eyes as tightly as he can. Note that the affected eye is either not closed at all—in which case the eyeball rolls upwards to make up for the failure of the lid to descend (*Bell's phenomenon*)—or, if the eye is closed, the eyelashes are not so much buried in the face as on the healthy side. Try also to open the eyes while the patient attempts to keep them closed. If the orbicularis is acting normally, it should be almost impossible to open them against the patient's wish.

The effect of screwing the eyes tightly shut causes the corners of the mouth to be drawn upwards. In paralysis of the lower part of the face, the corner on the affected side is either not drawn up at all, or not so much as on the healthy side.

2. Ask the patient to whistle. He is unable to do so.

3. Ask him to smile or show his upper teeth. The mouth is then drawn to the healthy side.

4. Ask him to inflate his mouth with air and blow out his cheeks. Tap with the finger in turn on each inflated cheek. Air can be made to escape from the mouth more easily on the weak or paralysed side.

5. Test the sense of taste on the anterior part of the tongue.

Signs of paralysis of the facial nerve in different parts of its course

Paralysis of the face presents different symptoms depending on whether the lesion is situated above the nucleus, at the nucleus or below it. The former constitutes upper motor neurone or *supranuclear facial paralysis*, the latter lower motor neurone or *infranuclear paralysis*.

The chief difference between the two forms is that in *supranuclear paralysis* the lower part of the face is chiefly affected; in *infranuclear paralysis* both the upper and lower parts are equally involved. This is because there is bilateral innervation of the muscles of the upper part of the face; a unilateral lesion, therefore, will only cause partial paralysis of one side. Sometimes a supranuclear lesion only affects the fibres concerned in emotional movement and this function should be tested separately from voluntary movement. Taste is not affected in supranuclear lesions.

Infranuclear facial paralysis may be produced by a lesion of the nucleus or of the facial nerve itself.

A lesion inside the facial canal—unless it is towards the outer end—involves the fibres of the chorda tympani and therefore produces loss of taste sensation in the anterior two-thirds of the tongue.

Lesions of the nucleus or the nerve below it will result in atrophy of the facial muscles. Supranuclear lesions do not produce this effect. Bilateral weakness of the face is sometimes difficult to detect.

Abnormal facial movements

The muscles supplied by the facial nerve are frequently affected by spasm or spasmodic movements. These may involve all the facial muscles, or groups of them only. The spasm may be either clonic or tonic (p. 251). If present, the nature of the movements, their extent, the muscles affected by them, and any factors which modify them should be carefully noted.

The eighth or vestibulocochlear nerve

Anatomy. This nerve consists of two sets of fibres. One supplies the cochlea and subserves the function of hearing; the other supplies the vestibule and semicircular canals and is the nerve of equilibration. The *auditory* fibres, which arise from the cochlear ganglion, enter the brain stem at the lower border of the pons and are distributed to the dorsal and ventral cochlear nuclei. The *vestibular* fibres take origin from the vestibular ganglion, and terminate in a group of nuclei in the pons and medulla.

The secondary auditory tracts, after partial decussation, terminate in the inferior colliculi and the medial geniculate bodies, and another system, that takes origin from these, passes through the internal capsule to the cortical centre for hearing, in the 1st and 2nd temporosphenoidal convolutions.

Sounds received in one ear reach the opposite hemisphere of the brain, but owing to the partial decussation of the secondary auditory tracts neither unilateral cerebral nor brain stem lesions produce deafness in one ear.

The vestibular nerve is closely connected with the cerebellum. It also has cerebral connections.

Tests of hearing

Before testing a patient's hearing, exclude the presence of wax in the ear (p. 281). This done, hearing can be tested with a watch. Ask the patient to shut his eyes. Begin outside the probable range of hearing and bring the watch gradually nearer the ear, asking the patient to speak when he hears the tick. It is necessary to know at what distance the tick should be audible to a healthy ear. Test each ear separately, keeping one closed with your finger tip whilst the other is being examined.

If impairment of hearing is detected, it must be determined whether it is really due to disease of the vestibulocochlear nerve or merely to some affection of the middle ear. In order to settle this point, the *tuning-fork* test may be employed. When the fork is sounding strongly, hold it opposite the ear; if it can be heard, then place its base on the mastoid process in order to determine if its vibrations can be heard when conducted through bone. If the patient hears this, ask him to compare the relative loudness of the fork when heard through air and through bone, or to determine which can be heard the longer as the vibrations die out. This is *Rinne's test*. Normally, aurally conducted sounds are louder to the patient than those conducted through bone. In middle ear disease, air conduction is diminished or lost, while bone conduction remains normal. When the auditory nerve is affected, both air and bone conduction are diminished or lost.

Weber's test, though less reliable than Rinne's, should also be used. Strike a tuning-fork and place the end of it against the centre of the patient's forehead. If the deafness discovered by the watch is due to an affection of the middle ear, the patient will hear the tuning-fork better on that side than on the healthy one. On the other hand, if the deafness is due to disease of the auditory nerve, the tuning-fork will only be heard on the healthy side. The test may also be carried out by means of the watch. In affections of the nerve, the watch is not heard even when pressed against the ear; in disease of the middle ear, it is heard even more loudly than when similarly applied to the healthy side.

These tests are frequently unreliable.

Other points in favour of the deafness being due to a lesion of the nerve and not to disease of the middle ear are: (a) if the hearing is better in a quiet place; (b) if conversation is heard better than the watch; and (c) if inflation of the middle ear renders the hearing worse.

Abnormal auditory sensations

The patient may complain of 'ringing in the ears' (*tinnitus*). The precise character of the sound varies in different cases. It may be of a humming, buzzing, hammering or whistling character. The presence or absence of this symptom should always be inquired for, and if present, whether it is constant, or in what circumstances it occurs. It is only rarely of diagnostic significance in neurological disease.

Hyperacusis, a disorder in which even slight sounds are heard with painful intensity, sometimes occurs when there is paralysis of the stapedius muscle due to a facial nerve palsy.

Hallucinations of sound may also be present, the patient fancying that he hears voices, bells etc. These occur chiefly in the psychoses, but they sometimes arise as epileptic aura when the causal lesion is situated in or near the auditory cortex.

Tests for vertigo

The patient will usually describe this as *giddiness* or *dizziness*. In order to constitute true vertigo, external objects should seem to move round him. Ask if this is the case and, if so, in what direction the objects seem to move. Ask also whether the vertigo causes him to fall to the ground. The distinction between giddiness and true vertigo is rarely of great importance. Both may occur in disease of the vestibular system and one should look, therefore, for signs of disease of the ear, 8th nerve or brain stem.

The ninth (glossopharyngeal), tenth (vagus) and eleventh (accessory) nerves

Anatomy. These arise, in order from above downwards, in an elongated nucleus in the floor of the 4th ventricle. They emerge by several roots along the lateral aspect of the medulla, beginning above in the groove between the olive and restiform bodies. The spinal part of the 11th emerges from the lateral column of the cord, beginning as low as the 6th cervical nerve; it passes up through the foramen magnum to join the medullary (accessory) part, and emerges with it through the jugular foramen. After emerging, its two divisions separate, the medullary or accessory portion joining the vagus and supplying motor fibres to the larynx and pharynx.

The glossopharyngeal nerve

The ninth (glossopharyngeal) is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle constrictor of the pharynx and for the stylopharyngeus. It contains the taste fibres for the posterior part of the tongue (p. 229).

The 9th nerve is rarely paralysed alone. Paralysis can best be diag-

nosed by examining its sensory and reflex functions. Examine taste in the posterior part of the tongue (p. 229). Loss of it may occur with a lesion of the trunk of the glossopharyngeal nerve.

Tickle the back of the pharynx, and note if reflex contraction occurs. This is also a test of the vagus (below).

The vagus nerve

The *tenth* (vagus) is motor for the soft palate (with the exception of the tensor palati), pharynx and larynx. It is sensory and motor for the respiratory passages, the heart and (through the parasympathetic ganglia) for most of the abdominal viscera. The fibres for the soft palate, pharynx and larynx take origin in the nucleus ambiguus, emerge in the upper roots of the 11th, reach the pharyngeal plexus, and thence pass to the muscles of the palate, the constrictors of the pharynx, and to the larynx. The visceromotor and the cardio-inhibitory fibres are derived from the dorsal vagus nucleus in the floor of the 4th ventricle.

Paralysis of the vagus is obvious clinically only through its palatine and laryngeal branches.

The palate. Ask the patient whether he is troubled with the regurgitation of fluids through his nose when he tries to swallow. This is a common occurrence in total paralysis of the soft palate, owing to defective elevation during swallowing. For a similar reason the patient is unable to pronounce words which require complete closure of the nasopharynx. Thus 'egg' is sounded as 'eng', 'rub' becomes 'rum', and so on. In unilateral paralysis these symptoms are not observed.

For direct examination of the soft palate, place the patient facing the light with his mouth open and introduce a tongue depressor. The position of the uvula is unreliable, as deviation is not uncommon even in health. One must watch the movements of the palate during phonation. Ask the patient to say 'Ah!' and observe whether both sides of the palate arch upwards. If one side is paralysed, it will remain flat and immobile and the median raphe will be pulled towards the other side. The manner in which the palate rises in such a case has been compared to the ascent of a curtain of which one string is broken. In bilateral paralysis the whole palate remains motionless.

Remember that minor degrees of asymmetry of the palate and of the tongue occur as part of a hemiparesis due to an upper motor neurone lesion. This is not what is meant by palatal palsy since in the latter the lesion is invariably of lower motor neurone type.

The larynx. The superior laryngeal branch of the vagus is sensory for the larynx above the level of the true vocal cords and is motor for the cricothyroid muscle. Unilateral paralysis of the nerve does not produce

any symptoms. Bilateral paralysis causes the vocal cords to be relaxed. The voice is therefore hoarse and deep and the utterance of high notes is impossible.

The recurrent laryngeal branch supplies sensation to the larynx below the level of the vocal cords and motor fibres to all the laryngeal muscles except the cricothyroid. Paralysis of it leads to appearances which can be recognized with the laryngoscope and which are described on p. 288. *The speech is characteristically blurred and ineffectual.* Bilateral paralysis may cause serious stridor or even respiratory obstruction.

The accessory nerve

The *eleventh* (accessory) nerve is purely motor in function, contributing to the innervation of the larynx and pharynx as well as the sternomastoid and trapezius. The spinal part of the nerve dips below the sternomastoid muscle about 2 cm below the tip of the mastoid process and re-emerges from underneath that muscle at about the middle of its posterior border. It supplies the sternomastoid and trapezius, which are also supplied by twigs from the cervical plexus.

Paralysis of the upper part of the trapezius is demonstrated by asking the patient to shrug his shoulders while the examiner presses downward on them. Paralysis of the sternomastoid causes weakness of rotation of the chin towards the opposite side.

The twelfth (hypoglossal) nerve

Anatomy. The twelfth nerve arises from a nucleus in the lower part of the floor of the fourth ventricle, close to the midline. It emerges between the anterior pyramid and the olive. It is a purely motor nerve, supplying the tongue and the depressors of the hyoid bone.

Test. Ask the patient to put out his tongue as far as possible. If the hypoglossal is paralysed the tongue, instead of being protruded straight, is pushed over to the paralysed side. Be careful not to mistake an apparent deviation of the tongue, really due to the mouth being twisted to one side, for a real deviation. Such an apparent deviation occurs in facial paralysis. Ask the patient to move his tongue from side to side, and to lick each cheek with it; observe whether he can do so freely. Strength may also be assessed by pressing against the tongue with a finger as the patient protrudes it into each cheek in turn. Note whether there is any wasting of the tongue, and whether there is any tremor or fasciculation of it. The presence of wasting indicates that the lesion is either nuclear or infranuclear (lower motor neurone).

MOTOR FUNCTIONS

The motor system should be examined with the following aspects of motor function in mind:

1. Bulk of muscles
2. Tone of muscles
3. Strength of muscles
4. Reflexes
5. Coordination of movement
6. Gait
7. Involuntary movements

Bulk of muscles

The bulk of the muscles is most easily estimated by inspection and palpation. Wasted or atrophic muscles are not only smaller, but also softer and more flabby, than normal when they are contracted. When muscular wasting is accompanied by fibrosis, as in muscular dystrophy or polymyositis, the muscles feel hard and inelastic; they become shortened and it is not possible to stretch them passively to a normal degree. *Contracture* is then said to be present. Contractures may also occur as a result of prolonged hypertonia in a group of muscles.

Muscular atrophy is not only caused by neurological disorders. Generalized muscular wasting is seen in cachexia of any cause. Localized muscle atrophy may be due to injury or disease of a joint; this occurs, for example, in the thenar muscles in association with arthritis of the 1st metacarpophalangeal joint or in the quadriceps in patients with diseases of the knee. In such instances strength is well preserved in relation to the degree of muscular wasting. Some patients with *muscular dystrophy* develop large muscles (pseudohypertrophy) due to pathological changes in the muscles themselves. The calves, buttocks and infraspinati are particularly affected. These enlarged muscles are weak in spite of their size.

Tone of muscles

Muscular tone is a state of tension or contraction found in healthy muscles. An increase in tone is spoken of as *hypertonia* and a diminution as *hypotonia*. The degree of tone is estimated by handling the limbs and moving them passively at their various joints. The maintenance of tone is dependent on a spinal reflex arc; afferent fibres from the primary and secondary endings of the muscle spindles entering the spinal cord and synapsing with the anterior horn cells, from which efferent fibres arise and pass to the muscles. Tone is diminished or lost if this reflex arc is damaged. *Hypotonia* therefore occurs in affections of the lower motor

neurone, of the afferent sensory pathways, as in tabes, and in cerebellar disease in which suprasegmental mechanisms are abnormal. Tone may be reduced in sleep and by certain drugs.

Muscle tone is also regulated by corticospinal and extrapyramidal pathways.

Hypertonia following lesions of the corticospinal system (upper motor neurone lesions) is termed *spasticity* and it has a characteristic distribution; the upper limbs are held in flexion and the lower limbs in extension with the feet in plantar flexion (physiological extension). Spasticity is a term which should only be used to describe a state of increased tone which is of 'clasp-knife' type when the limb is fairly rapidly flexed or extended. These are the lengthening and shortening reactions described by Sherrington. Spasticity is therefore a form of rigidity which is *stretch sensitive*. Moreover it can usually be shown that the degree of increased tone developed during any given passive stretch is velocity-dependent, i.e. it is proportional to the speed of the applied stretch.

Hypertonia resulting from disease of the basal ganglia is termed *extrapyramidal rigidity*. The hypertonia is more uniform, but often it is so distributed as to produce a general attitude of flexion of the limbs and trunk as in Parkinson's disease. The resistance to passive movement is regularly or irregularly variable and is aptly described as like a lever rubbing on the teeth of a cogwheel (cogwheel rigidity). Sometimes a plastic type of rigidity is found in which the resistance developed to passive movement is uniform during all phases of the applied movement. This is often referred to as *paratonic rigidity* or '*Gegenhalten*'. It is found in catatonic states, in patients with clouded or confused intellects from any cause and, commonly, in dementia. It should not be regarded simply as evidence of 'lack of cooperation'. Its physiological basis is unknown. In *hysterical rigidity* the resistance to passive movement increases in proportion to the effort applied by the examiner. The increased resistance is usually developed in a characteristically jerky fashion.

When the muscles are *hypotonic*, there is little or no resistance of passive movement of the limb and when handled or shaken the unsupported part flops about inertly. Hypotonic muscles are abnormally soft to palpation. The outstretched hypotonic upper limb usually assumes an abnormal posture, as in cerebellar disease or chorea. It is hyperextended at the elbow, the forearm is over pronated, the wrist usually flexed and the fingers hyperextended at the metacarpophalangeal joints.

Strength of muscles

Much the quickest and most reliable method of making a quick or preliminary assessment is to watch the patient walking, jumping, hopping,

standing up from lying and sitting positions and dressing or undressing. These movements require proximal and distal strength and coordination of a considerable degree and much can be learnt by observing them carefully. The strength of individual muscles can then be assessed as required. Each movement made during this assessment is tested by comparison with the examiner's own strength or by comparison with what he judges to be normal in a person of comparable build to the patient. It therefore requires practice and experience. It will be found that very simple requests produce better results than long explanations: a demonstration or gesture is often more effective than any verbal explanation, if the patient does not immediately understand a request. Remember that most patients have no knowledge of anatomy.

Testing the muscles of the upper limb

Abductor pollicis brevis. This is an important muscle as it is supplied by the median nerve which is commonly damaged by compression in the carpal tunnel at the wrist. The patient is asked to abduct his thumb in a plane at right angles to the palmar aspect of the index finger. The muscle can be seen and felt to contract.

Opponens pollicis. Ask the patient to touch the tip of his little finger with the point of his thumb.

First dorsal interosseous. Ask the patient to abduct his index finger against resistance.

Interossei and lumbricales. Test the patient's ability to flex his metacarpophalangeal joints and to extend the distal interphalangeal joints. The interossei also adduct and abduct the fingers. When these muscles are paralysed and power is retained in the long flexors and extensors of the fingers, a claw-hand or deformity is produced. The first phalanges are overextended and the distal two are flexed. The fingers are slightly separated.

Flexors of the fingers. Ask the patient to squeeze your fingers. Allow him to squeeze only your index and middle fingers—this is sufficient to assess strength of grip without his painfully crushing your fingers.

Flexors of the wrist. Ask him to bring the tips of his fingers towards the front of the forearm.

Extensors of the wrist. Ask the patient to make a fist, a movement which results in firm contraction of both the flexors and extensors of the wrists, and try to forcibly flex the wrist against his effort to maintain his posture. It should be almost impossible to overcome the wrist extensors of a healthy man or woman. The wrist flexors can be similarly tested.

Slight weakness of the extensors of the wrist may be elicited by asking the patient to grasp something firmly in his hand. If the extensors are weak the

wrist becomes flexed as he does so, because the flexor muscles are then stronger than the extensors.

Weakness or paralysis of the extensors of the wrist as in radial nerve palsy leads to *wrist-drop*.

Brachioradialis. Place the arm midway between the prone and supine positions; then ask the patient to bend up the forearm, whilst you oppose the movement by grasping the hand. The muscle, if healthy, will be seen and felt to stand out prominently at its upper part.

Biceps. Ask the patient to bend up the forearm against resistance, with the forearm in full supination. The muscle will stand out clearly.

The *triceps* is tested by asking the patient to straighten out his forearm whilst the observer endeavours to keep it flexed.

Supraspinatus. Ask the patient to lift his arm straight out at right angles to his side. The first 30° of this movement is carried out by the supraspinatus. The remaining 60° is produced by the deltoid.

Deltoid. The anterior and posterior fibres help to draw the abducted arm forwards and backwards respectively.

Infraspinatus. The patient is asked to tuck his elbow into his side with the forearm flexed to a right angle. He is then asked to rotate the limb outwards against the examiner's resistance, the elbow being held against the side throughout. The muscle can be seen and felt to contract.

Pectorals. Ask the patient to stretch his arms out in front of him, and then to clap his hands while the observer endeavours to hold them apart.

Serratus anterior. When this muscle is paralysed the scapula is 'winged', the vertebral border projecting. The patient is unable to elevate his arm above a right angle, the deformity becoming more apparent as he tries to do so. Pushing forwards with the hands against resistance, such as a wall also brings out the deformity.

Latissimus dorsi. The patient is asked to clasp his hands behind his back while the observer, standing behind the patient, offers passive resistance to the downward and backward movement; alternatively the two posterior axillary folds can be felt as the patient coughs.

Testing the muscles of the trunk

Weakness of the muscles of the abdomen is shown by the patient's inability to sit up in bed from the supine position without the aid of his arms. *Babinski's 'rising up sign'* consists in making the patient lie on his back with the legs extended and rise up without using his hands. In organic spastic paralysis of a leg the affected limb will rise first, owing to the rigidity, but in hysterical paralysis this does not occur.

Paralysis of a portion of the anterior abdominal wall can be detected by the displacement of the umbilicus that occurs when the patient attempts to lift up his head from the pillow against resistance. With paralysis of the lower segment the umbilicus moves upwards, but when the upper segment is affected the umbilicus is pulled downwards. This is sometimes known as *Beevor's sign*: it is a useful sign since it may indicate the level of a lesion in spinal cord disease.

To test the *erector spinae* and muscles of the back, make the patient lie on his face and try to raise his head from the bed by extending the neck and back. If the back muscles are healthy, they will be seen to stand out prominently during this effort.

The method of detecting paralysis of the *diaphragm* has already been described (p. 42).

The *trapezius* is tested in its upper part by asking the patient to shrug his shoulders while the observer tries to press them down from behind. In its lower part it can be tested by asking him to approximate the shoulder blades.

The *cranial musculature* has already been described (p. 230).

Testing the muscles of the lower limb

The *intrinsic muscles of the foot* cannot be easily examined. When the interossei are weakened or paralysed a 'claw-foot', analogous to the 'claw-hand', may develop. Rarely this deformity occurs in patients with spastic hemiparesis of very long duration; this is due to a form of dystonia and not to muscular weakness.

Dorsiflexion and *plantar flexion* of the feet and toes are tested by asking the patient to elevate or depress the part against resistance.

Eversion and *inversion* of the foot should be investigated by instructing the patient to turn the foot outwards or inwards against resistance.

Extensors of knee. Bend up the patient's knee, and then, pressing with your hand on his shin, ask him to try to straighten it out again.

Flexors of knee. Raise the leg up from the bed, supporting the thigh with your left hand, holding the ankle with your right. Then ask the patient to bend his knee.

Extensors of the thigh. The knee being extended, lift the patient's foot off the bed, and ask him to push it down against resistance. If the extensors of the hip are paralysed he will be unable to do so.

Flexors of the thigh. With the leg extended ask the patient to raise his leg off the bed against resistance.

The *adductors of the thigh* are tested by abducting the limb and then asking the patient to bring it back to the middle line against resistance. In a similar way the *adductors* are tested by bringing the limb across the middle line and then asking the patient to move it outwards again.

Rotators of the thigh. With the lower limb extended on the bed, ask the patient to roll it outwards or inwards against resistance.

Weakness (*paresis*) is often graded according to a *Medical Research Council* scale as:

Grade 0	Complete paralysis
Grade 1	A flicker of contraction only
Grade 2	Power detectable only when gravity is excluded by appropriate postural adjustment
Grade 3	The limb can be held against the force of gravity, but not against the examiner's resistance
Grade 4	There is some degree of weakness, usually described as poor, fair or moderate strength
Grade 5	Normal power is present

The term *hemiplegia* is applied to a condition in which there is paralysis of one side of the body, especially of the arm and leg, and usually also of the face. If the paralysis of the arm and leg is on one side, and that of some of the muscles supplied by the motor cranial nerves on the other, the condition is one of *crossed paralysis*. This is a sign of disease in the brain stem. The term *paraplegia* is applied to a paralysis of both legs; the term *monoplegia* to a paralysis of one limb, which may be the arm (*brachial monoplegia*) or the leg (*crural monoplegia*). In *quadriplegia* all four limbs are weak.

The detection of hemiplegia in a patient who is comatose is often very difficult. However, if the paralysis is of recent onset, one can usually detect hypotonia in the paralysed limbs. If his arm, for example, is raised from his side and allowed to drop, it falls, if it is paralysed, as if it did not belong to him; the sound arm also falls, but not in such an utterly limp fashion. The face is asymmetrical, the angle of the mouth more open on the paralysed side, and the affected cheek moves loosely outwards and inwards with respiration. The abdominal and tendon reflexes may be absent on both sides, but an extensor plantar response can usually be obtained on the hemiplegic side (see Reflexes, p. 242).

Myasthenic weakness

In *myasthenia gravis* weakness, which commonly affects the external ocular and bulbar muscles more than the rest of the skeletal musculature, is characteristically exacerbated or provoked by prolonged contraction or exercise of the affected muscles. The degree of detectable weakness therefore varies during the course of the day. This is referred to as *myasthenic weakness*. A similar form of weakness in which there is an initial increase of strength with contraction, occurs as a rare phenomenon in carcinomatous neuromyopathies.

Myotonia

In certain myopathies and muscular dystrophies, of which *dystrophia myotonica* is the best known, relaxation after contraction of muscle is impaired. The phenomenon is worse when the affected muscles are cold and it is therefore often best demonstrated in the hand. Ask the patient

to grip your hand firmly and then let go *suddenly*. His grasp is maintained for a moment and then is slowly and gradually released. Subtle degrees of myotonia can be demonstrated in the tongue and in other muscles, e.g. the thenar eminence, by lightly striking the muscle with a small patellar hammer. A dimple of contraction appears and relaxes only slowly.

Reflexes

Tendon reflexes

If the tendon of a lightly stretched muscle is struck a single, sharp blow with a soft rubber hammer (thus suddenly stretching the muscle and exciting a synchronous volley of afferent impulses from the primary sensory endings of the muscle spindles in the stretched muscle), the muscle contracts briefly. This is the monosynaptic stretch reflex. It is a test of the integrity of the afferent and efferent pathways, and of the excitability of the anterior horn cells in the spinal segment of the stretched muscle. Properly performed, examination of the tendon reflexes offers a reliable and reproducible method of assessment of the integrity of this system of neurones and their higher connexions, and it is therefore very important to become skilled in the techniques for eliciting these reflexes.

Always use the same type of hammer; always examine these reflexes in the same manner, standing on the same side of the bed: always make sure the patient is warm and comfortable and reassure him that the hammer is soft and not an offensive weapon. When examining the tendon reflexes in the legs care taken to allow the patient's genitalia to be properly covered is repaid by more easily elicitable reflexes. The patient should be asked to relax or to 'let the muscles go to sleep'.

Knee jerk. The knee jerk is the best known of the tendon reflexes. The patellar hammer derives its name from its invention as an instrument for eliciting this reflex, the first tendon reflex to become a regular part of the neurological examination. The knee jerk consists of a contraction of the quadriceps when the patellar tendon is tapped. The spinal segments concerned are the 2nd, 3rd and 4th lumbar. It is best tested with the patient supine. The examiner's hand is passed under the knee to be tested and placed upon the opposite knee; the knee to be tested rests on the dorsum of the observer's wrist. The patellar tendon is struck midway between its origin and insertion. Following the blow there will be a brief extension of the knee from contraction of the quadriceps. The reflex can sometimes be more easily elicited with the patient sitting up, his legs dangling freely over the edge of the bed.

The briskness of the knee jerk varies greatly in different individuals. In health it is hardly ever entirely absent. Sometimes, as in the case of

the other tendon reflexes, one is unable to elicit it without applying *reinforcement* (Jendrassik's manoeuvre). This is done by asking the patient to make some strong voluntary muscular effort with the upper limbs; for example to hook the fingers of the two hands together and then to pull them against one another as hard as possible. While he is doing this, a further attempt is made to elicit the knee jerk. Reinforcement acts by increasing the excitability of the anterior horn cells, and by increasing the sensitivity of the muscle spindle primary sensory endings to stretch (by increased gamma fusimotor drive).

Ankle jerk. Place the lower limb on the bed so that it lies everted and slightly flexed. Then, with one hand, slightly dorsiflex the foot so as to stretch the Achilles tendon and, with the other hand, strike the tendon on its posterior surface. A sharp contraction of the calf muscles results. This reflex can also be conveniently elicited when the patient is kneeling on a chair. It depends upon the 1st and 2nd sacral segments.

Triceps jerk. Flex the elbow and allow the forearm to rest across the patient's chest. Tap the triceps tendon just above the olecranon. The triceps contracts. The reflex depends upon the 6th and 7th cervical segments. Care must be taken to strike the triceps *tendon* and not the belly of the muscle itself. All muscles show a certain amount of irritability to direct mechanical stimuli; but this is a direct response, not a stretch reflex.

Biceps jerk. The elbow is flexed to a right angle and the forearm placed in a semipronated position; the examiner then places his thumb or index finger on the biceps tendon and strikes it with the patellar hammer. The biceps contracts. The 5th and 6th cervical segments of the cord are concerned.

Supinator jerk. A blow upon the styloid process of the radius stretches the supinator causing supination of the elbow. This reflex depends on the 5th and 6th cervical segments.

With lesions at this level the supinator reflex may be lost but, when it is tested, brisk flexion of the fingers is seen. This phenomenon is known as *inversion* of the reflex and it is evidence of hyperexcitability of the anterior horn cells below the C5/6 level. The responsible lesion is in the C5/6 spinal segments.

Jaw jerk. Ask the patient to open his mouth, but not too widely. Place one finger firmly on his chin and then tap it suddenly with the other hand as in percussion. A contraction of the muscles that close the jaw results. This jerk is sometimes absent in health and is increased in upper motor neurone lesions above the 5th nerve nuclei.

Clonus. The phenomenon of clonus is often elicitable when the tendon reflexes are exaggerated as a result of a pyramidal lesion.

Ankle clonus. Bend the patient's knee slightly and support it with one hand, grasp the forepart of the foot with the other hand and suddenly dorsiflex the foot. The sudden stretch causes a reflex contraction of the calf muscles which then relax, but continued stretch causes a regular oscillation of contraction and relaxation which is called *clonus*. When it is *sustained* it is evidence of an upper motor neurone lesion and it is then always associated with increased tendon reflexes (hyperreflexia) and an extensor plantar response. *Unsustained clonus* may occur in healthy persons, particularly in those who are very tense or anxious.

Grading the reflexes. The tendon reflexes may be graded:

- 0 Absent
- 1 Present (as a normal ankle jerk)
- 2 Brisk (as a normal knee jerk)
- 3 Very brisk
- 4 Clonus

Abnormal tendon reflexes. The tendon reflexes are diminished or absent with lesions affecting the afferent pathways, the anterior horn cells themselves, or the efferent pathways (lesions of the lower motor neurone). For example, in tabes dorsalis the posterior roots are affected; in poliomyelitis the anterior horn cells are diseased; and in most peripheral neuropathies both the efferent (motor) and afferent (sensory) nerve fibres are abnormal; in all these conditions certain tendon reflexes may be absent.

Hyperreflexia occurs with upper motor neurone lesions (at all levels above the anterior horn cells). It may also occur with anxiety or nervousness, in thyrotoxicosis and as a manifestation of tetanus. Hyperreflexia is therefore only of pathological significance if it is asymmetrical or if it is associated with other signs of an upper motor neurone lesion (spasticity, weakness or an extensor plantar response).

In cerebellar disease the reflexes may have a characteristic *pendular* quality. This is clearly evident only when there is a severe cerebellar ataxia and it is not a sign of diagnostic importance. It may be considered a manifestation of hypotonia.

In myxoedema both the contraction and relaxation phases of the tendon reflex may be prolonged. The relaxation time can be estimated by simple observation with surprising accuracy in relation to the normal and is a sensitive and reliable clinical index of hypothyroidism. This is sometimes called a *myotonic reflex*.

Superficial reflexes

The plantar reflex. Assessment of the plantar reflex is of great clinical importance, not least because it is an objective response which can be easily compared by various observers. To elicit it the muscles of the lower limb should be relaxed. The *outer edge of the sole of the foot* is stimulated by gently scratching a key or a pin along it from the heel towards the little toe and then medially across the metatarsus. In healthy adults even a slight stimulus produces a contraction of the tensor fascia lata, often accompanied by a slighter contraction of the adductors of the thigh and of the sartorius. With a slightly stronger stimulus, flexion of the four outer toes appears, which increases with the strength of the stimulus till all the toes are flexed on the metatarsus and drawn together, the ankle being dorsiflexed and inverted. This is called the *flexor plantar response*. With still stronger stimuli withdrawal of the limb occurs. It is doubtful that the plantar reflex is ever completely absent in healthy subjects.

Babinski's sign. In lesions of the corticospinal system an abnormality in the plantar response was first described by Babinski. Extension of the great toe precedes all other movement. It is followed by spreading out and extension of the other toes, dorsiflexion of the ankle and flexion of hip and knee. The small amount of movement at the ankle is less conspicuous than the brisker movement of the normal response.

This *extensor plantar response* (so called because the movement described is extension according to anatomical terminology, although the reflex is, in reality, part of the nociceptive flexion withdrawal response described in the decerebrate preparation by Sherrington), is most easily elicited by stimulation of the *outer part of the sole*. With slight corticospinal lesions, it may be evoked from this region alone when a normal flexor response is obtained by stimulating the medial edge of the sole. If the lesion is progressive, the area in which the extensor plantar reflex can be excited (receptive field) increases and spreads first inwards and over the sole of the foot, and then upwards along the leg to the knee or even the groin. For this reason extension of the great toe, generally associated with some dorsiflexion of the foot, can sometimes also be obtained by squeezing the calf or pressing heavily along the inner border of the tibia (*Oppenheim's sign*), or by pinching the calcaneus tendon (*Gordon's reflex*).

In adults, the extensor plantar response occurs only in cases of disease involving corticospinal pathways, but in children below the age of 1 year the extensor response is the normal response. The flexor response appears in the subsequent 6–12 months as myelination of the corticospinal pathways is completed.

TABLE 8. CHIEF SUPERFICIAL REFLEXES OF SPINAL ORIGIN

<i>Reflex</i>	<i>How excited</i>	<i>Result</i>	<i>Level of cord concerned</i>
Anal	Stroking or scratching the skin near the anus	Contraction of the anal sphincter	3rd and 4th sacral segments
Bulbocavernosus	Pinching dorsum of glans penis	Contraction of bulbo-cavernosus	3rd and 4th sacral segments
Plantar	Stroking sole of foot	Movements of toes, of toes and foot, or leg	Lower part of lumbar enlargement (5th lumbar and 1st sacral segments)
Cremasteric	Stroking skin at upper and inner part of thigh*	Drawing upwards of testicle	1st and 2nd lumbar segments
Abdominal	Stroking abdominal wall below costal margin, at level of umbilicus and in iliac fossa	Contraction of abdominal muscles	7th to 12th thoracic segments
Scapular	Stroking skin in interscapular region	Contraction of scapular muscles	5th cervical to 1st thoracic segment

* The cremasteric reflex can often be most easily elicited by pressing over the sartorius in the lower third of Hunter's canal.

Flexor spasms may occur during testing of the plantar reflex. These consist of an exaggerated extensor plantar response, the whole limb being suddenly drawn up into flexion and the large toe extended. This is the fully developed human counterpart of Sherrington's nociceptive flexion withdrawal response. It is common in spinal cord disease and in some patients with bilateral upper motor neurone lesions at a higher level. Flexor spasms are often particularly severe in the presence of posterior column disease (as in multiple sclerosis or subacute combined degeneration), or when there is a constant stimulation of small unmyelinated fibre input to the spinal cord, as in the presence of bedsores or urinary tract infection in a patient with a cord lesion. *Extensor spasms*, conversely, are more likely to occur when posterior column function is normal.

Superficial abdominal reflexes. These are elicited by allowing the patient to lie, relaxed and in a supine position, with the abdomen uncovered. A light stimulus, such as a key or a thin wooden stick, is passed across the abdominal skin in the plane of the dermatome from the outer aspect towards the midline. A ripple of contraction of the underlying abdominal musculature follows the stimulus. These reflexes are absent in upper motor neurone lesions above their spinal level. In disease of the thoracic spine they may indicate the segmental level of the lesion by their absence below this level.

It is often impossible to obtain them in anxious patients, in the elderly or obese and in multiparous women.

Corneal reflex. See p. 229.

Palatal reflex. See p. 234.

Sphincteric reflexes

This term includes the reflexes necessary for swallowing, micturition and defaecation. They depend upon complex muscular movements excited by increased tension in the wall of the viscus concerned.

One should ascertain from the patient whether he has any difficulty in swallowing (*dysphagia*), noting especially whether there is any regurgitation of food through the nose. As a rule patients with neurological disorders causing dysphagia complain of difficulty in swallowing liquids, whereas those with mechanical oesophageal obstruction complain they cannot swallow solids.

Defaecation. The patient should be questioned as to any difficulty and as to the presence of rectal sensation. Incontinence of faeces should be noted.

The reflex action of the anal sphincter may be tested by introducing the oiled, gloved finger into the anus and noting whether contraction of

the sphincter occurs with normal force, whether it is weak or paralysed, or whether any spasm is excited. The activity of the reflex may also be tested by gently pricking the skin near the anus. A brisk contraction of the sphincter should immediately occur.

Micturition. The patient should be questioned as to difficulty in controlling or initiating micturition and as to whether bladder and urethral sensation are normal. Retention and incontinence or urgency of micturition should be noted.

Incontinence in neurological disorders may be due to overflow from an atonic distended bladder in which sensation has been lost. In this case the bladder will be enlarged to palpation or percussion and suprapubic pressure may result in the expulsion of urine from the urethra. Incontinence may also be due to *reflex incontinence*, either occurring at regular intervals as the bladder partially fills, or precipitately and unexpectedly in response to a sudden noise, to movement or to exposure to cold.

Coordination of movement

By coordination is meant the smooth recruitment, interaction and cooperation of separate muscles or groups of muscles, in order to accomplish a definite motor act. If such coordination is imperfect, motor performance becomes difficult or impossible and *ataxia* is said to be present.

The coordination of groups of muscles is the product of various factors, among which are the afferent impulses coming from the muscle spindles and joint receptors, cerebellar function and the state of tone of the muscles. When ataxia is present it is not always easy to say which of these factors is at fault. The movements that constitute an act can be controlled and directed by vision, but sight itself is not concerned in the coordination of most normal movements. When, however, there is loss of the sense of position of a limb or joint, the sensory defect may be compensated for by vision, and the disturbance of movement may become apparent only when the eyes are closed or the patient is in the dark. Such ataxia occurs typically in *tabes dorsalis*, when position sense is diminished or lost in the legs. In any patient with ataxia therefore, it is important to ascertain whether joint position sense is impaired or not before the ataxia can be ascribed solely to cerebellar disease. Proximal weakness may mimic cerebellar ataxia, but this can usually be distinguished easily when muscular strength is tested.

How to test coordination

In the upper limbs. Ask the patient to touch the point of his nose first with one forefinger and then with the other; or ask him to touch first

his nose, then the examiner's forefinger with his own index finger. If he performs these movements naturally and without making random errors no incoordination is present. He may then be asked to perform the same actions with his eyes closed; any additional irregularity of the movements can be due only to disturbance of the sense of position.

It is often useful to watch the patient dressing or undressing, handling a book or picking up pins.

In the lower limbs. If the patient is able to walk, a good test in the lower limbs consists of asking him to walk along a straight line. If incoordination is present he will soon deviate to one side or the other.

If he cannot walk, ask him, as he lies in bed to lift one leg high in the air, to place the heel of this leg on the opposite knee and then to slide the heel down his shin towards the ankle. In cerebellar ataxia a characteristic, irregular, side to side series of errors in the speed and direction of movement occurs. The test should be performed *with the eyes open*.

Another method is to ask the patient to describe a circle in the air with his toe or forefinger. If he is able to describe circles accurately coordination is good.

A special and very useful sign of cerebellar ataxia is *dysdiadokokinesia*; it consists of inability to execute rapidly repeated movements. The patient is asked to flex his elbows to a right angle and then alternately to supinate and pronate his forearms as rapidly as possible 'as though screwing in a light bulb'. All normal persons can do this very rapidly but usually, slightly less rapidly with the left than with the right arm. When, however, dysdiadokokinesia is present the movements are slow, awkward and incomplete, and often become impossible after a few attempts. The rhythm of the movement becomes characteristically irregular. The sign can also be elicited by asking the patient to tap the examiner's palm with the tips of his fingers as fast as possible. Minor degrees of ataxia can then be both felt and heard.

Romberg's sign is often regarded as a special test for the coordination of the lower limbs. It is, however, a test for loss of position sense or sensory ataxia in the legs and not a test of cerebellar function. However, patients with severe aural vertigo or cerebellar ataxia may also show some instability in the test.

The patient is asked to stand with his feet close together and if he can do this, he is then asked to close his eyes. If Romberg's sign is present, as soon as his eyes are closed he begins to sway about or may even fall. *The essential feature of the sign is therefore that the patient is more unsteady standing with his eyes closed than when they are open.* In the presence of sensory ataxia, as in *tabes dorsalis*, the patient is unable to maintain his posture without the aid of vision on account of defective position sense in his legs.

Gait

The character of a patient's gait is often important in diagnosis, particularly in cases of neurological disease. The legs must be fully exposed. The patient should therefore either be unclothed or wear underpants. The feet should be bare. The patient is asked to walk away from the observer, to turn round at a given point and then to come towards him again.

The points to be noted are:

1. Can the patient walk at all? If he can:
2. Does he walk in a straight line or does he tend to deviate to one side or the other? To bring out this point, ask him to walk along a straight line, e.g. a crack in the floor.
3. Does he tend to fall and, if so, in what direction?

The next point to be decided is whether the gait conforms to any of the well recognized abnormal types. Before deciding this, *be quite sure* that the peculiarity in the patient's gait is not due to some surgical cause or to local disease of a joint, e.g. osteoarthritis of the hip. Examination of the bones and joints will eliminate such sources of error.

Common disorders of gait due to neurological disease are:

Spastic gait. The patient walks on a narrow base, has difficulty in bending his knees and drags his feet along as if they were glued to the floor.

The foot is raised from the ground by tilting the pelvis, and the leg is then swung forwards so that the foot tends to describe an arc, the toe scraping along the floor.

The gait is seen most characteristically in patients with pyramidal lesions. The *hemiplegic gait* is essentially a spastic gait in which only one leg is affected.

The gait in *sensory ataxia* may be described as 'stamping'. The patient raises his feet very suddenly, often abnormally high, and then jerks them forward, bringing them to the ground again with a stamp and often heel first. If he watches the ground, he may be fairly steady, as he can use his eyes in place of his position sense, but he becomes severely ataxic when his eyes are closed or if he walks in the dark. This gait is best seen in *tabes dorsalis*. Other signs of loss of postural sensibility will be present.

The gait of *cerebellar incoordination* may be described as a 'drunken' or 'reeling' gait and requires no further description. Patients with this gait walk on a broad and irregular base, the feet being planted widely apart. The ataxia is equally severe whether the eyes are open or closed.

This gait occurs in disease of the cerebellum or cerebellar tracts and other signs of ataxia will be present.

Festinant gait. This occurs in Parkinson's disease. The patient is bent forwards, and advances with rapid, short, shuffling steps, so that 'he looks as if he were trying to catch up with his centre of gravity', and his arms do not swing. In some cases, if he is suddenly pulled backwards, he begins to walk backwards, and is unable to stop himself (*retropulsion*).

The *waddling gait* is like the gait of a duck. The body is usually tilted backwards, with an increase of lumbar lordosis; the feet are planted rather widely apart and the body sways from side to side as each step is taken. The heels and toes tend to be brought down simultaneously. This gait disorder is due to difficulty maintaining truncal and pelvic posture because of proximal muscular weakness. It occurs, therefore, in the myopathies and the muscular dystrophies. A similar gait may occur with bilateral disease of the hip joints (Trendelenberg's sign).

The *high-stepping gait* is a device adopted by the patient to avoid tripping from his toes catching the ground. It occurs when there is weakness of the extensor muscles of the feet for example in lateral popliteal nerve palsy.

Involuntary movements

Involuntary, unintended movements occur, either at rest or during voluntary movement, in a number of different diseases of the nervous system. The different clinical varieties of involuntary movement are not specific disease entities, but represent clinical patterns of involuntary movement observed in many patients. Most are due to diseases of the basal ganglia and extrapyramidal system.

Epilepsy

The possibility that an involuntary movement limited to one side of the body, or to one limb, might be due to focal epilepsy should always be considered. Very rarely such an attack may continue for hours or even days (*epilepsia partialis continua*). The movement is usually complex and repetitive. It may be exacerbated by arousal or by handling or touching the limb and it will usually be relieved by anticonvulsant drugs. The movement differs from the involuntary movement disorders principally by its stereotyped repetitiveness.

Myoclonus

Myoclonus is a rapid, irregular jerking movement of the limb or of the whole body, often occurring in response to extraneous stimuli, such as a sudden loud noise. A sudden start when surprised, or the bodily jerks which occur on falling asleep or waking, are common varieties of myoclonus experienced by most normal people. In exaggerated form these jerks of *flexion myoclonus* may

occur as a manifestation of major epilepsy or in some patients with degenerative disorders of the cerebellum. Generalized flexion myoclonus also occurs in certain types of encephalitis, when it often exhibits an obvious periodicity. Less commonly, irregular myoclonic jerks may occur in a single limb or a ripple of jerky irregular contraction may pass through the muscles of a limb. Myoclonus can occur with lesions at many levels in the nervous system and it does not, therefore, have localizing value.

Tremor

Regular or irregular distal movements having an oscillatory character are classified as tremors. The tremor of *anxiety* is fine and rapid. It is similar to the tremor found in *thyrotoxicosis*. Coarser distal tremor, often exaggerated in awkward postures, as when the outstretched fingers are held pointing at each other in front of the patient's nose and usually relieved to some extent during movement, occurs as a familial disorder: *benign essential tremor*. This tremor is often coarse, but usually irregular, and it is present both at rest and during movement. It must therefore be distinguished from parkinsonian and cerebellar tremors. The latter is present only during movement (*intention tremor*) and the former is accompanied by signs of extrapyramidal disease. *Senile tremor* is similar to benign essential tremor. *Hysterical tremor* tends to involve a limb or the whole body and it is, characteristically, worsened by the examiner's attempt to control it.

The tremor of *parkinsonism* is usually easily recognizable. It consists of a rapid rhythmic alternating tremor, predominantly in flexion/extension but often with a prominent rotary component between finger and thumb (*pill-rolling tremor*). More proximal muscles may be involved, and the lips and tongue are frequently affected. The tremor is invariably more severe in the arm than in the leg. It is often strikingly unilateral and is invariably associated with other symptoms and signs of extrapyramidal disease, such as hypokinesia, cogwheel rigidity, postural abnormalities and gait disorder.

Athetosis

Athetosis is a writhing movement, usually more pronounced in distal than in proximal muscles, in which the play of movement is very complex but often seems to consist of a relatively constant interaction between two postures, those of *grasping and avoiding*. The fingers are alternately widely extended, the arm following into an extended, abducted and externally rotated posture, and then the fingers clench, often trapping the thumb in the palm, and the limb flexes slightly and internally rotates. In very severe forms of this disorder, as for example in *dystonia musculorum deformans* (torsion dystonia) the trunk and axial musculature is also affected and the patient may scarcely be able to stand.

Athetosis may be unilateral or generalized. The latter form is usually associated with degenerative disease of the basal ganglia. Very rarely it may occur as a paroxysmal phenomenon.

Chorea

This word means 'a dance'. The involuntary movements are brief, fluid and often difficult, at first to discern. Ordinary voluntary movements, such as walking or picking up a cup and saucer, may be embellished with smooth, rapid extra little flourishes of movement. Muscular tone is often decreased. The outstretched upper limbs may assume a hyperpronated posture and little flicks of movement of the digits or wrist may occur. At rest the patient appears 'fidgety' and 'unable to sit still'. The movements often appear less obvious during voluntary movement, and are increased by agitation or nervousness. Chorea occurs as part of an inherited presenile dementia (*Huntington's chorea*), associated with rheumatic fever (*Sydenham's chorea*) and rarely in pregnancy and after certain drugs (phenothiazines). It may also occur with other systemic diseases, e.g. thyrotoxicosis and systemic lupus erythematosus, and in old age (*senile chorea*). Unilateral chorea may occur with deeply placed lesions in one hemisphere.

Dyskinesias

In the past this word was reserved for *phenothiazine-induced* involuntary movements, which particularly affect the pharyngeal peri-oral musculature, and also for *levodopa-induced* axial torsional movements. Latterly the word has come to be used simply to describe any involuntary movement.

Dystonia

This word is used to describe an abnormally maintained posture, often associated with a plastic rigidity. The dystonias are closely related to choreo-athetosis, but the term can also be used to describe the flexed posture of Parkinson's disease (flexion dystonia) or the hemiplegic posture (hemiplegic dystonia).

Hemiballismus

This is a peculiar and unique involuntary movement, almost invariably unilateral and affecting the arm more than the leg, in which the limb is flung rapidly, and often with great force, from full extension into abduction and external or internal rotation. The movements may be so violent as to result in serious injury to the limb and to loss of weight. The disorder is associated with lesions in the region of the subthalamic nucleus.

Tics

These are simple normal movements which become repeated unnecessarily to the point that they become an embarrassment or a source of or reaction to

psychiatric problems. They can be readily imitated in contrast to the other involuntary movement disorders. Head nodding is a common example.

Myokymia

This is a persistent twitchy and often rhythmical movement of the periorbital muscles. It may occur as a benign phenomenon in fatigued or anxious people. It is sometimes due to lesions in the facial nerve or its nucleus.

Metabolic flap (asterixis)

An irregular, abrupt, brief loss of posture, especially evident in the outstretched hands or tongue occurs in decompensated hepatic failure (*hepatic flap*) and in other metabolic disorders, e.g. uraemia, poisoning with hypnotic drugs, and in respiratory failure.

Tetany

Tetany, commonly due to hypocalcaemia or alkalosis, can be recognized by the characteristic posture of the affected hand (*main d'accoucheur*). Ischaemia of the affected limb, produced by a sphygmomanometer cuff inflated above the arterial pressure, will augment this sign or produce it if it is not already present (*Trousseau's sign*). Another useful test is to tap lightly with a patellar hammer in the region of exit of the facial nerve from the skull, about 3–5 cm below and in front of the ear. The facial muscles twitch briefly with each tap (*Chvostek's sign*).

SENSATION

The following different forms of sensation are usually tested:

1. *Tactile sensibility*. This includes light touch and pressure, and tactile localization and discrimination.
2. *Position sense*, and the appreciation of passive movement.
3. *Recognition of the size, shape, weight and form of objects*.
4. *Appreciation of vibration*.
5. *Pain*.
6. *Temperature*.

The presence or absence of any abnormal sensations should be noted.

These 'sensory modalities' do not necessarily represent different, discrete sensory functions but, rather, are commonly experienced sensations in normal life. Perception depends on a complex physiological interaction of afferent input at many levels in the nervous system and, in some instances as in the recognition and naming of objects, it depends also on the ability to manipulate the object felt. Perception of vibration, for example, does not depend on a special set of nerve fibres responsible only for transmitting vibration sense to the central nervous system, but rather is a form of sensation subjectively similar to light touch which, in clinical practice is found to be disturbed when there is a lesion of the large diameter, afferent fibres in the peripheral nerves, posterior columns or, more rarely, at a higher level than this.

Begin testing sensation with touch and position sense. Use a pin later, when you have gained the patient's confidence. Always apply the sensory stimulus first to an area of impaired sensation and mark out its borders *from the abnormal to the normal*. The patient should readily note the sudden change to normality.

Areas of diminished sensation should be carefully, *but quickly*, mapped out so that their distribution in relation to root lesions, peripheral nerve lesions or lesions in the central nervous system can be studied. It is important to do this quickly, accurately and, as far as possible, without repetition. The longer the time spent on this the more confusing will be the result: it requires great concentration and cooperation from the patient and from the examiner.

Inconsistency in the patient's replies may be due to fatigue, poor cooperation, dementia or undue suggestibility. This can be checked by asking the patient to say 'now' whenever he feels the stimulus, his eyes being shut; by examining a related form of sensation such as temperature sense in the case of pinprick, or position or vibration sense in the case of light touch; or by exhorting the patient to be very careful to make the correct response. Never make much of small differences. Remember that many patients will experience changes in the acuteness or sharpness of a pin between the nail bed and the dorsum of the finger, or at about the level of the clavicle when the stimulus ascends the anterior chest wall. The pulp of the fingers is rather insensitive to pain, but very sensitive to light touch and discriminative tests such as two point discrimination. Vibration sense is best perceived over a bony prominence. On the whole it is better to test sensation with the patient's eyes open. During most sensory testing it should not affect the result to have the patient actually watching the procedure. It can be a frightening experience to be suddenly pricked with a pin when one's eyes are closed and such surprises, which destroy a patient's confidence in the examiner, should always be avoided.

Tactile sensibility

Use a wisp of cotton wool or the tip of your index finger.

Ask the patient to indicate whether he feels the touch, and if it feels normal. If not, *how* is it abnormal? It may be abolished or reduced (*hypoaesthesia*), misperceived as a painful, irritating or tingling sensation (*hyperaesthesia*) or mislocalized. Very rarely there may be a delay between the stimulus and its recognition by the patient. Areas of diminished sensation should be carefully delineated and recorded.

Ability to discriminate between two points is tested by the use of blunt dividers. The patient is asked whether he is being touched with one or both points. Normally 2 mm of separation of the points can be recognized as two separate stimuli on the finger tips, and slightly more

separation on the pulps of the toes. This is an excellent objective sensory test which is particularly useful in cases of posterior column or parietal cortical lesions; and in some peripheral nerve lesions (such as the carpal tunnel syndrome).

Position sense

Ask the patient to look away or shield his eyes. Explain that you will move his finger (or toe or elbow) up or down and ask him to tell you which way it has been moved. He should be able to recognize movements of only a few degrees at all joints, including knee, ankle, elbow and wrist, in addition to the more commonly tested fingers and toes. It is sometimes helpful to ask the patient to imitate with the opposite limb or digit the position of the limb or digit being tested. It is essential that the patient be relaxed and that he allows the limb to be moved *passively*.

When the position sense is disturbed in the upper limbs, the outstretched fingers may twist, rise and fall when held with the eyes closed. These involuntary movements (pseudo-athetosis) occur unknown to the patient and disappear almost completely when the patient watches the position of his fingers. Patients with defective position sense may be unable to manipulate small objects, fasten buttons and so on without visually observing their movements (sensory ataxia).

The *appreciation of movement* is closely related to the sense of position and can be tested at the same time. Gradually move a digit or limb into a new position, with the patient's eyes closed and ask him to say 'now' as soon as he recognizes the movement. Note the angle through which the limb was moved. If the appreciation of movement is diminished this angle is many times greater than that in a normal limb. Movements of less than 10° can be appreciated at all normal joints. Finally test that the patient can recognize the direction of the movement, that is, whether the joint is flexed or extended. Patients can sometimes recognize the occurrence of a movement but not its direction.

Recognition of size, shape and form

These faculties can be tested most accurately in the hands with the eyes closed. To test size, place in the patient's palm objects of the same shape, but of different sizes, for example small rods or matches of different length. Ask him to say which is the larger. The objects should be applied consecutively.

To test recognition of shape familiar objects such as coins, a pencil, a pen-knife, scissors, etc. are placed in the hand, and the patient is asked to identify them or to describe their form. Loss of this faculty is known as *astereognosis*. It may occur, with parietal lesions, when position sense and light touch are normal, although there is usually some defect in these modalities. When astereognosis occurs with posterior column lesions position sense, vibration sense and light touch are invariably profoundly disturbed.

Appreciation of vibration

If the foot of a vibrating tuning-fork is placed on the surface of the body the vibrations can be felt, provided they are sufficiently strong. This is a valuable test, as the ability to appreciate vibration may be lost in various diseases, as in *tabes dorsalis*, in peripheral neuritis and in posterior column disorders. A tuning fork of 128 Hz (middle C) should be used. Vibrations of higher frequency are more difficult to perceive. If the patient perceives the vibration ask him to say when he ceases to feel it. If the examiner can then still perceive it, the patient's perception of vibration is impaired. There is often some loss of vibration sense in the feet and legs in old age.

Pain

Pain may be evoked either by a cutaneous stimulus as the prick of a pin, or by pressure on deeper structures, such as muscles or bones. Sensibility to superficial and to pressure pain should be tested separately.

Superficial pain. The point of a pin should be used as the stimulus. Care must be taken that the patient distinguishes between the *sharpness* of the point (that is, its relative size) and the *pain* which the prick evokes; it often happens that, even when sensibility to pain is abolished, he can recognize that the stimulus is pointed, and thus confuse the observer by calling it 'sharp'.

Pressure pain is examined by squeezing the muscles or the Achilles tendon. Abolition of pressure pain is often the most prominent sensory disturbance in *tabes dorsalis*.

Absence of sensibility to pain is termed *analgesia*; partial loss of pain sensibility is called *hypoalgesia*; and an exaggerated sensibility, so that even a mild stimulus causes an unnatural degree of painful sensation, is known as *hyperalgesia*. This occurs in some patients with spinal cord disease, for example in *tabes dorsalis*, and in certain patients with deep-seated parietal or thalamic lesions (thalamic pain). The pain experienced has a peculiar, ill-localized and persistent character. It often has a burning quality and it may occur as an intractable spontaneous phenomenon or only in response to cutaneous stimuli.

Temperature sense

Temperature sense is conveniently examined by using test tubes containing warm and cold water. The part to be tested is touched with each in turn, and the patient says whether each tube feels hot or cold.

Other disturbances of sensation

Sensory inattention. This phenomenon is sometimes found in patients with lesions of the parietal lobe; it is demonstrated as follows. The patient is asked to close his eyes. Homologous points on opposite sides of the body are stimulated simultaneously by touch or with pins. The patient is asked to indicate which side, or sides are touched and, in the presence of sensory inattention he fails to perceive the stimulus on the abnormal side. This is sometimes called *sensory extinction*. *Bilateral simultaneous sensory stimuli* can also be studied when testing vision and hearing and a similar defect may be found. In the presence of hemisensory loss, of course, the sign is invalid.

Some patients with parietal lesions will also show *spatial summation*, a sensory abnormality in which a stimulus is only perceived if an area of skin larger than a certain critical area is stimulated, or *temporal summation* in which an ill-localized and often perverted or painful sensation is felt after rapidly repeated stimuli. Single stimuli will be missed. These abnormalities are part of a perceptual defect related to sensory agnosia, a disorder in which the patient is unaware of the nature or the severity of his sensory disorder. In its most extreme form there may even be denial of illness (*anosagnosia*). In patients with higher perceptual defects of this type a number of other bedside tests may be useful. These include *constructional tests* such as the patient's ability to draw a map of his surroundings, to copy a complex figure (for example, two interlocking, irregular pentagons), to draw a clock face or a human face, or to draw more complex figures, for example a house. Visual and tactile memory can be tested by variations of these tests. Constructional ability is particularly impaired with right parietal lesions (*constructional apraxia*).

SIGNS OF MENINGEAL IRRITATION

Neck stiffness

The patient is asked to flex his neck as fully as he can to ascertain the degree of movement possible, and then to relax. The examiner then passively flexes the neck. The chin should normally touch the chest without pain.

In meningeal irritation the test causes pain in the neck, sometimes radiating down the back, and the movement is resisted by spasm in the extensor muscles of the neck. Neck rigidity is also caused by diseases of the cervical spine. Head retraction is an extensive degree of neck rigidity.

Kernig's sign

Kernig's sign is tested by passively extending the patient's knee when his hip is fully flexed. This movement causes pain and spasm of the

hamstrings in meningeal irritation affecting the lower part of the spinal subarachnoid space. It is a less sensitive test than neck rigidity.

These two tests depend upon the fact that stretching the spinal nerve roots in conditions of meningeal irritation causes a reflex muscular spasm. They are positive in meningitis and subarachnoid haemorrhage, but also in patients with 'meningism', a state of irritation of the meninges, seen most commonly in young children with acute fevers; and in some patients with raised intracranial pressure in whom herniation of the cerebellar tonsils into the foramen magnum has begun.

Straight leg raising

This test is used in patients with sciatica. The sciatic nerve and its roots are stretched by passively elevating the patient's extended leg with the hand, which is placed behind the heel. The movement is restricted by pain in conditions in which the spinal roots are involved as in protrusion of a low lumbosacral intervertebral disc.

SPECIAL INVESTIGATIONS

The following special methods of investigation are in common use.

Lumbar puncture

This is a procedure used for obtaining samples of cerebrospinal fluid (CSF) which requires some experience. It should be carried out under supervision in the first instance. It is performed as follows:

Mark out the 3rd and 4th lumbar spines. The 4th lumbar spine usually lies in the plane of the iliac crests. The puncture may be made through either the 3rd or 4th interspace. The patient should be lying on his side on a firm couch, with the knees and chin as nearly approximated as possible. His back should be right at the edge of the couch and it is *important that its transverse axis*, i.e. a line passing through the posterior superior iliac spines, *should be vertical*. Local anaesthesia may be produced by injecting 2% sterile procaine, first raising a bleb under the skin, and, when this is insensitive, anaesthetizing the whole dermis. It is not necessary to inject procaine into the deep ligaments. This procedure usually causes more pain than it relieves. A special needle about 8 cm in length should be used. The stylet should fit accurately and should not protrude through the bevelled cutting edge of the needle.

Push the needle firmly through the skin in the middle line or just to one side of it and press it steadily *forwards and slightly towards the head*, the bevel pointing towards the side on which the patient is lying. When the needle is felt to enter the spinal cavity the stylet is withdrawn and the CSF which escapes is collected in 3 sterilized stoppered test tubes.

If any blood is present, a marked difference in the amount in the first and subsequent tubes indicates that the blood is due to trauma from the puncture. The patient should lie flat for 8 to 24 hours afterwards.

It is sometimes useful to have a manometer connected with the needle, so that the pressure of the fluid can be measured at the time of puncture. If this is done, the patient's head must be on the same level as the sacrum and he must be breathing quietly and with his muscles relaxed. The neck and legs should be slightly extended. The normal pressure is from 60 to 150 mm of fluid.

Queckenstedt's test can be used to detect a block to the circulation of fluid in the spinal canal.

With the needle and manometer in position and the patient breathing quietly as described above, an assistant compresses one or other, but not both, jugular veins. This causes a sudden increase in intracranial pressure, which is immediately seen in the manometer as a sudden rise of cerebrospinal fluid pressure, followed by an equally rapid fall when the pressure on the vein is released. A similar sudden rise and fall is seen if the patient is asked to cough and this is a useful check that the needle tip is in free communication with the subarachnoid space.

With slight degrees of block there may be a rise of pressure in the manometer followed by a very slow fall when the pressure on the vein is released; and with more severe block no rise of pressure will be seen when the jugular vein is compressed.

This test is now rarely used since it has largely been superseded by myelography. *It should never be carried out in the presence of raised CSF pressure* since it may then precipitate transtentorial or tonsillar herniation.

Much the commonest cause of a 'dry-tap', the failure to obtain CSF, is an incorrectly performed puncture, and this is usually due to the patient not being in the correct position. The needle will then not be introduced at right angles to the transverse axis of the back, and will miss the spinal canal. Occasionally, however, a 'dry-tap' is due to a complete block to the flow of CSF through the spinal canal. In this circumstance urgent myelography is required and for this procedure *cisternal puncture* may be required. This should only be performed by an experienced physician or surgeon. It is best undertaken under radiographic control.

Lumbar puncture should *never* be performed in patients in whom raised intracranial pressure is suspected. The fundi should *always* be examined before a lumbar puncture is performed in order to exclude papilloedema.

Abnormalities of the fluid

Normal cerebrospinal fluid is clear and colourless like water. Any yellowness is pathological and is due either to old haemorrhage,

jaundice or excess of protein. In *Froin's syndrome* a pronounced yellow colour (xanthochromia) is associated with great excess of protein and the formation of a coagulum. It is a very rare phenomenon. Even slight increases in CSF protein, however, cause a noticeable increase in viscosity of the fluid when it is gently shaken, and an excessive frothiness of its surface.

Turbidity of the fluid may be due to the presence of white blood cells, either as a result of infection or of subarachnoid haemorrhage. If it does not clear on standing it is due to micro-organisms.

The presence of *blood* may be due to injury to a vessel by the needle or to subarachnoid haemorrhage. In the latter case the blood is more uniformly mixed with the fluid, and the supernatant fluid, after centrifugation, is yellow.

Cytological examination of a turbid fluid is of great importance. A centrifugal deposit should be examined with Leishman's stain in order to obtain an idea of the character of the cells present; and by Gram's and Ziehl-Neelsen's methods for bacteria. Cell counts are performed with the aid of a counting chamber (p. 141) and must be done immediately the fluid has been collected. Counts done some hours later give inaccurate results because the leucocytes stick together and to the sides of the tube, and endothelial cells break up in a short time. If any clot has formed an accurate cell count cannot be obtained but the cells in the clot can be stained and examined. Normal fluid contains 2-5 lymphocytes/ μ l.

An excess of cells ('*pleocytosis*') is described as being of polymorphonuclear type, if these cells are above 75% of the total and of the lymphocytic type if more than 90% are lymphocytes. Bacterial meningitis is associated with a polymorphonuclear pleocytosis, virus meningitis and syphilis with a lymphocytic one, and tuberculous meningitis and poliomyelitis with either a lymphocytic or a mixed type.

The CSF should also be examined bacteriologically (p. 338) and chemically.

Normal cerebrospinal fluid contains only a trace of albumin and hardly any globulin, the *total protein* being not more than 40 mg/100 ml. In some neurological diseases, particularly in multiple sclerosis and in many acute and subacute virus infections, the globulin fractions in the cerebrospinal fluid are altered. The *Lange test* takes advantage of this. Varying dilutions of cerebrospinal fluid are mixed in ten tubes with a colloidal gold suspension of constant strength. The degree of precipitation which results is expressed by arbitrary figures 0-5, 0 representing no change and 5 complete precipitation. The CSF IgG concentration can also be directly estimated by immunoelectrophoresis and in many laboratories this estimation has replaced the Lange test.

Glucose is present in normal cerebrospinal fluid in a concentration of

50–75 mg/100 ml, which is about a half to a third of the blood glucose concentration. In purulent, tuberculous or fungal meningitis and rarely in carcinomatous meningitis the CSF sugar is reduced to less than half of the blood glucose.

One or more of the tests for syphilis (p. 331) are often performed on the cerebrospinal fluid.

The typical changes in the CSF in various neurological diseases are summarized in Table 9.

The electroencephalogram (EEG)

Electrodes applied to the patient's scalp pick up small changes of electrical potential, which after amplification are recorded on paper. The EEG is of particular value in the investigation of epilepsy and in the localization of cerebral tumours and other expanding intracranial lesions. It is also a useful research technique.

The electromyogram (EMG)

Electrical activity occurring in muscle during voluntary contraction, or in denervated muscle during rest, can be recorded with needle electrodes inserted percutaneously into the belly of the muscle, or with surface electrodes (silver discs attached to the skin overlying the muscle with a salty paste), amplified and displayed as an auditory signal through a suitable loudspeaker and as a visual signal on a cathode ray oscilloscope. They may be recorded on magnetic tape or by photography. Analysis of such electrical activity is useful in the diagnosis of primary diseases of muscle (*myopathies* and *dystrophies*) and of lower motor neurone lesions (denervation).

The speed of conduction of afferent impulses (*sensory nerve conduction velocity*) and efferent impulses (*motor nerve conduction velocity*) in peripheral nerves can be estimated using an electrical nerve stimulation technique and suitable recording electrodes and amplifying or digital averaging equipment. These measurements are useful in the diagnosis of peripheral nerve disorders, particularly those due to local compressive lesions as, for example, carpal tunnel syndrome.

Neuroradiology

Apart from routine radiographs of the skull and spine a number of *contrast techniques* are useful.

Myelography (Plate XVIII)

This is a method for demonstrating the subarachnoid space in the spinal canal. A lumbar or cisternal puncture is performed with the patient on the myelogram table in the X-ray department and 2–10 ml of a special

TABLE 9. TYPICAL CHANGES IN THE CEREBROSPINAL FLUID IN VARIOUS DISEASES

Disease condition	Physical characteristics	Cytology	Protein (mg/100 ml)	Glucose (mg/100 ml)	Tests for syphilis	Stained deposit	Culture	Range curve
Normal	Clear and colourless	0-5 cells/ μ l	10-40	45-90*	Negative	No organisms	Sterile	0000000000 0000110000
Meningitis								
Bacterial	Yellowish and turbid	Polymorphs 200-2000 Lymphocytes 5-50	50-200	0-15	Negative	Bacteria	Positive	0001344310
Tuberculous	Colourless sometimes viscous	Polymorphs 0-100 Lymphocytes 100-300	50-200	15-50*	Negative	Tubercle bacilli in films in some cases	Positive by special methods	0001344310
Viral (includes poliomyelitis)	Usually clear	10-100 mixed cells at first, becoming lymphocytic in 36 hours	30-60 Poliomyelitis 100-200, remaining high 6-8 weeks	45-90	Negative	No organisms	Sterile	0001344310
Multiple sclerosis	Clear and colourless	Rarely 5-15 lymphocytes	30-60, rarely higher	45-90	Negative	No organisms	Sterile	Paretic type of curve in 50% of cases
Syphilis								
GPI	Clear and colourless	5-100 lymphocytes	40-100	45-60	Positive	No organisms	Sterile	5555432100
Tabes	Clear and colourless	5-100 lymphocytes	30-60	45-70	20% negative by reagin tests†	No organisms	Sterile	0123210000
Meningeal	Clear and slightly turbid	10-50 polymorphs 50-500 lymphocytes	50-200	May be slightly low	Positive	No organisms	Sterile	0123210000

* CSF glucose is usually about half the blood glucose. Simultaneous blood and CSF glucose estimations should always be performed.
† Nearly all are positive by FTA-ABS or TPHA.

radiopaque contrast medium or 10–25 ml of oxygen or air (radiolucent or negative contrast) are injected into the subarachnoid space. By tilting the table the contrast medium can be made to flow up and down the spinal canal under direct vision, preferably using TV amplification; and radiographs can be taken of regions of deformity or obstruction to the flow of contrast. This is a very useful method for accurate localization of tumours in the spinal canal.

Angiography (Plate XVIII)

This is a method for studying the intracranial and extracranial vessels. A suitable radiopaque contrast medium is injected percutaneously into a carotid artery in the neck, or into a vertebral artery either by direct puncture or by catheterization of a major vessel such as the femoral, axillary, brachial or subclavian arteries. Radiographs are taken in various planes in the following few seconds as the contrast medium traverses the cerebral circulation. The arterial, capillary and venous circulations can be studied and abnormalities of the distribution, size, position and lumen of these vessels can be seen.

The technique is particularly useful for diagnosis of aneurysms, arteriovenous malformations and cerebral tumours. It is less useful in cerebral vascular disease. General anaesthesia is usually preferred.

Air encephalography (AEG) (Plate XVII)

5–30 ml of air or oxygen, injected as in myelography by lumbar puncture, are allowed to flow from the spinal canal into the subarachnoid cisterns of the posterior fossa, thus outlining the cerebellar hemispheres and brain stem. By judicious positioning of the head, it is then made to enter the ventricular system. In this way the fourth ventricle, the aqueduct and the third and lateral ventricles can be studied. A series of anteroposterior, postero-anterior, lateral and oblique radiographs are taken, often using tomographic techniques and a very accurate demonstration of the anatomy of the brain itself can be obtained. It is sometimes necessary to supplement AEG by the additional instillation of 2–5 ml of radiopaque contrast medium (Myodil encephalography).

Air encephalography may be dangerous in the presence of raised intracranial pressure and in these patients, as in many of those with hydrocephalus, the injection of air or Myodil may be made through frontal or parietal burr holes, directly into the ventricles (*air* or *Myodil ventriculography*). With increasingly accurate angiographic technique this investigation is less often necessary now than formerly.

Very careful positioning of the head is required for air encephalography and special mechanical chairs are used in many departments to facilitate this. It is usual therefore to perform the investigation under general anaesthesia.

Diagnostic ultrasound

A pulsed ultrasonic beam is directed through the head in a lateral direction from one temporal region to the other. Reflections are recorded by a second transducer on the same side of the head. The position of the ultrasonic midline can be compared with that of the third ventricular walls and, occasionally, of the walls of the lateral ventricles themselves. The technique requires practice but it is particularly useful in the management of patients unconscious after head injuries. Unlike the radiographic procedures described above it is 'non-invasive' and can be repeated as often as necessary.

Computer-assisted axial tomography (EMIs can)

This is a newly devised method of obtaining tomographic sections of the brain of very high resolution, without the need for contrast procedures. A crystallographic X-ray detection device is used instead of conventional X-ray film and a photographic picture is produced by computerized averaging techniques. The method promises to reduce the need for air encephalography and angiography.

ROUTINE EXAMINATION OF THE NERVOUS SYSTEM

A detailed examination of the whole nervous system is time-consuming and something of an ordeal for the patient. There follows a scheme for a quick routine examination, which will be found useful in the examination of patients not suspected of neurological disease: and will also indicate what functions require detailed examination in those with such disease.

Mental state

Much useful information can be obtained during history taking and physical examination; no specific questions need usually be asked.

Is the history given *accurately, concisely* and *with insight*? Or is the patient *concrete, circumlocutory* or *vague*?

Is his *memory* normal?

Is he *neatly dressed* and *well cared for*?

Is his *behaviour* normal?

Is he *aphasic* or *dysarthric*?

Is he *confused*?

Can he *find his way* in and out of the room?

Can he *dress and undress* himself?

Gait

Is it *spastic, hemiparetic, ataxic* or *parkinsonian*?

Is there a *foot drop*?

Cranial nerves

Test *ocular movements*, looking for nystagmus.

Test *facial movements*.

Test *tongue protrusion* and *palatal movement*.

Visual fields

These, like aphasia, may provide absolute evidence of disease above the tentorium cerebelli.

Is there a *hemianopia*?

If so, is it *homonymous*, *bitemporal*, *unilateral* or something else?

Is *central vision* normal? This is crudely assessed by testing the visual acuity. Can he read small print with or without glasses?

Fundi

Is *papilloedema* present?

Is *optic atrophy* present?

Are there *hypertensive*, *uraemic* or *diabetic* changes present?

Motor

Is there any *weakness* of the outstretched upper limbs?

Is there *distal* or *proximal weakness* or *wasting*?

Is *muscular tone* normal, spastic or extrapyramidal in type?

Look for *cerebellar ataxia* in the limbs.

Assess the *tendon reflexes* and *plantar responses*.

Sensory

Test *position sense* in the fingers and toes and vibration sense in the feet (posterior columns).

Test *pin-prick* in the four limbs and on the face (lateral spinothalamic tracts).

Light touch need only be tested, if the patient complains of numbness.

General

Examine the *skull*, *spinal movements* and *posture*.

Look for *cutaneous naevi*.

Listen for *bruits* in the neck.

10

EXAMINATION OF THE EYE, EAR, NOSE AND THROAT

*The Eye — The Ear — The Larynx — The Nasal Part of the Pharynx — The
Nose — The Nasal Sinuses*

THE EYE

Before examining the various parts of the eye, one should test the visual acuity, and, when relevant, the colour sense and the extent of the visual fields.

Visual acuity

Test for distant vision

Visual acuity is measured with Snellen's test-types, a series of letters of varying sizes so constructed that the top letter is visible to the normal eye at 60 metres, and the subsequent lines at 36, 24, 18, 12, 9, 6 and 5 metres respectively. Visual acuity (V) is recorded according to the formula $V = d/D$, where d is the distance at which the letters are read, and D that at which they should be read. The patient is normally placed at a distance of 6 metres from the test types ($d = 6$) and each eye is tested separately. The patient reads down the chart as far as he can. If only the top letter of the chart is visible, the visual acuity is 6/60. A normal person should be able to read at least the seventh line, i.e. a visual acuity of 6/6. A person with an uncorrected refractive error may have a subnormal visual acuity, and a rough estimate of his corrected visual acuity may be obtained by asking him to view the chart through a pin-hole aperture. If the visual acuity is less than 6/60, the patient is moved towards the test-types until he can read the top letter. If the top letter is visible at 2 metres, the visual acuity is 2/60. Visual acuities of less than 1/60 are recorded as 'counting fingers' (CF), 'hand movements' (HM), 'perception of light' (PL) or 'no perception of light' (no PL).

If the patient wears glasses, the type of lens he is wearing may be determined as follows. Hold the lens in front of the eye and look at an object through it. Then move the lens from side to side and watch the object. If the latter seems to move in the opposite direction to the lens, the lens is convex; if in the same direction, it is concave. Patients with myopia use concave (diverging) lenses and those with hypermetropia convex (converging) ones.

In order to tell whether a lens is spherical or cylindrical, look at a straight object through it and then slowly twist the lens round. If the lens is cylindrical, the object will appear to take up an oblique position. Patients who are astigmatic need cylindrical lenses.

Test for near vision

Visual acuity at the ordinary reading distance is assessed with reading test types of varying sizes, the notation being based on the printers' 'point' system. The smallest print used is N5. The near vision is recorded as the smallest type which the patient can read comfortably.

Colour sense

This is most easily tested by the use of pseudo-isochromatic plates, the best known being those of Ishihara. People with defective colour vision confuse certain colours. Pseudo-isochromatic plates are so constructed that a person with normal colour vision will read one number on a plate, while a person with defective colour vision will read a different number on the same plate.

The most common anomalies of colour vision are various types of red-green deficiency, inherited as sex-linked recessive conditions, which occur in about 8% of males and 0.5% of females in this country. People with blue-yellow deficiencies and with total colour-blindness are rare.

Visual fields

The testing of the visual fields is described on p. 212.

Examination of the eye

After the visual functions have been tested, the eyes should be examined systematically. The shape and position of the eyelids should be noted. Mongolian races have a long narrow palpebral aperture with an upward and outward obliquity and a characteristic fold of skin along the upper lid. The highest point of the aperture is at the junction of its middle and inner thirds, whereas in mongolism (Down's syndrome) the oblique palpebral aperture is short and wide with its highest point at the centre of the lid.

Ptosis, or drooping of the upper lid, may be congenital or acquired. A congenital ptosis may be unilateral or bilateral, whereas an acquired ptosis is usually unilateral if due to paralysis of the third cranial nerve (p. 220) or of the cervical sympathetic (p. 227), and usually bilateral if due to myopathy.

Lid retraction is present if a band of white sclera is visible above the

iris when the eyes are looking straight ahead. Lid retraction, which is usually due to thyrotoxicosis, is often associated with infrequent blinking and with lid-lag, i.e. the upper lid seems to lag behind the eyeball when the patient looks downwards. Patients with thyrotoxicosis frequently also have a slight degree of forward displacement of the eyeball (exophthalmos). Some patients with thyrotoxicosis or some whose thyrotoxicosis has been successfully treated medically or surgically, develop a more severe and progressive form of exophthalmos, which may be associated with ocular palsies. Exophthalmos also results from space-occupying lesions in the orbit, and an apparent exophthalmos may be present when the eye is longer than normal, as in myopia.

The presence of an inflammation of the margins of the lids (marginal blepharitis) should be noted, together with any abnormality in the position of the lid margins, i.e. eversion (ectropion) or inversion (entropion) of the lashes.

The *lacrimal gland* is examined by pulling up the outer part of the upper lid while asking the patient to look downwards and inwards. Acute inflammations (dacryoadenitis) result in a tender swollen gland with oedema of the upper lid and localized conjunctival injection. Chronic dacryoadenitis, a painless enlargement of the lacrimal gland which is frequently bilateral, occurs in the reticulosos, sarcoidosis and tuberculosis. Tumours of the lacrimal gland produce a hard swelling of the gland associated with proptosis.

The *conjunctiva* lining the eyeball (bulbar conjunctiva) and that lining the inner surface of the eyelids (palpebral conjunctiva) should be examined. In order to examine the palpebral conjunctiva of the lower lid, the lower lid should be pulled down while asking the patient to look upwards. To expose the palpebral conjunctiva of the upper lid, ask the patient to look downwards, then place the right thumb at the upper part of the upper lid and pull it upwards so as to evert the eyelashes. Grasp the lashes between the forefinger and thumb of the left hand and evert the lid by rotating it round the right thumb. The conjunctiva may be pale in anaemia, jaundiced, or injected in conjunctivitis. Marked injection of the bulbar conjunctiva with a mucopurulent discharge suggests a severe bacterial inflammation; marked injection with a little serous discharge is indicative of a viral infection; slight oedema of the conjunctiva with a milky hue suggests an allergic condition. Follicles on the upper palpebral conjunctiva occur in trachoma, whereas their presence on the lower palpebral conjunctiva suggests an allergic condition or a conjunctivitis of viral origin.

In *conjunctivitis* the injection is maximal in the fornices (the junction of bulbar and palpebral conjunctiva), and this appearance must be distinguished from the circumcorneal injection that occurs in keratitis, anterior uveitis and acute glaucoma. In circumcorneal injection there is

a narrow band of dilated blood vessel around the limbus, and the injection is minimal in the fornices.

Inflammations of the cornea (keratitis) may be superficial or deep, and are accompanied by circumcorneal injection. Superficial keratitis and corneal ulcers result in breaches in the corneal epithelium, and these breaches will stain with fluorescein. A drop of fluorescein is instilled into the conjunctival sac and the excess dye is then washed out with normal saline. Breaches in the corneal epithelium are stained green. Deep keratitis results in a hazy cornea, often with an intact epithelium; it is usually caused by a viral or syphilitic infection. Both keratitis and trauma to the cornea may result in corneal opacities; small opacities are described as nebulae, larger ones as leucomata.

Arcus senilis is a crescentic opacity near the periphery of the cornea. It usually starts at the lower part of the cornea, extending to form a complete circle. It is common in old people, but may occur in the young (*arcus juvenilis*). It does not appear to have any significance.

In *anterior uveitis* (iridocyclitis) circumcorneal injection also occurs. In addition, white specks may be visible on the posterior surface of the cornea (keratic precipitates); there may be an exudate in the anterior chamber, and the pupil may be constricted and irregular due to the formation of adhesions between the edge of the pupil and the anterior surface of the lens (posterior synechiae). Other abnormalities of the pupils are described on p. 226.

The *ocular tension* may be roughly assessed by palpating the eyeball, although even with practice only gross variations from normal can be appreciated. The sclera is palpated with the two forefingers through the upper lid with the patient looking downwards, the other fingers resting on the patient's forehead. The degree of fluctuation gives an indication of the ocular tension. More accurate measurements of ocular tension can be made with Schiøtz or applanation tonometers. A diminished tension occurs in diabetic coma and in severe dehydration from any cause. A myopic eye frequently feels softer than a normal one. An increased ocular tension occurs in glaucoma.

The fundus

Examination of the fundus of the eye with an ophthalmoscope is an essential part of every complete medical examination. Valuable information may be obtained as to the state of the optic nerve head and of the arteries and veins of the retina, in addition to the detection of local ophthalmic conditions.

In routine medical examinations it is usually possible, with practice, to examine the optic disc and surrounding retina without dilating the pupil, but for a complete examination of the fundus the pupils should be dilated by instilling a few drops of 1% cyclopentolate (Mydrilate) or

2% homatropine into the conjunctival sacs. In patients with a predisposition to closed-angle glaucoma an acute attack may be precipitated when the pupils are dilated. Before a mydriatic is instilled, the patient should be asked whether he has even seen haloes (coloured rings) around lights, and, if he has, this, or the presence of a shallow anterior chamber, is a contraindication to the use of a mydriatic. After the examination of the fundus has been completed, the effects of the mydriatic should be counteracted by the instillation of a few drops of 2% pilocarpine.

The patient should be examined either sitting or lying down in a darkened room. He is asked to look straight ahead and to keep his eyes as still as possible. The ophthalmoscope is held a few centimetres from the patient's eye, and a suitable plus lens is used in the ophthalmoscope so that the iris is in focus. Opacities in the media of the eye (cornea, anterior chamber, lens, vitreous) will appear as black specks or lines against the red reflex of the fundus.

The ophthalmoscope should then be brought as close as possible to the patient's eye and the light directed slightly nasally. In this way the optic disc can be found, and in addition the light will not shine directly on the macula. If the patient's pupils are not dilated, shining a light on to the macula will make the pupils contract and may make the examination of the fundus difficult or impossible. If the optic disc is not in focus, the strength of the lenses of the ophthalmoscope should be gradually reduced until the disc becomes sharply focused. If the observer's eye is emmetropic and his accommodation is relaxed, the strength of the lens necessary to bring the fundus into focus gives an indication of the refractive error of the patient's eye. Plus lenses indicate hypermetropia, and minus ones myopia. The optic disc, the retinal blood vessels, the macular region and the periphery of the fundus should be examined in turn.

The optic disc

Shape. The normal disc is round or slightly oval. If astigmatism is present, the disc may appear more oval than normal.

Colour. The normal disc has a pale pink colour, distinctly paler than the surrounding fundus. The temporal side of the disc is usually paler than the nasal side.

In atrophy of the optic nerve, the disc becomes very pale and may even become white or greyish-white in colour. In oedema of the optic nerve-head, resulting from raised intracranial pressure (papilloedema) or from inflammation (papillitis), the disc is pinker than normal and may approach the colour of the surrounding retina. In pseudopapilloedema, a congenital anomaly usually associated with hypermetropia,

the disc may appear swollen and pinker than normal, but the retinal blood vessels are normal in appearance, corrected vision is normal and the condition is stationary.

Physiological cup. In its central part there is usually a depression in the disc, the physiological cup. The cup is paler than the surrounding disc, and from it the retinal vessels enter and leave the eye. In glaucoma the cup may be greatly increased in size, and the retinal vessels will kink as they cross the edge of the disc. When the cup is deep (in advanced glaucoma), retinal vessels disappear as they climb from the floor to the rim, and reappear again as they bend sharply over the edge of the cup (Plate XIII).

Edge of the disc. This is normally well defined. In normal eyes there is sometimes a white scleral ring, a dark pigmented ring, or a stippled choroidal ring surrounding the optic disc.

The retinal blood vessels. These radiate from the disc, dividing dichotomously into many branches as they pass towards the periphery of the retina. The retinal arteries are narrower than the veins, are a brighter red in colour, and have a brighter longitudinal streak where light is reflected from their convex walls. Spontaneous retinal artery pulsation is an abnormal finding, and occurs in some cases of glaucoma and aortic regurgitation. Spontaneous venous pulsation is frequently seen in normal eyes; it never occurs in papilloedema. It is important to study the points where arteries and veins cross. Most frequently it is the artery that crosses the vein, and in normal eyes neither vessel shows any change in colour, diameter or direction.

The macular region. This is situated about 1.5 disc diameters from the temporal border of the optic disc. It is recognized by being darker in colour than the surrounding fundus, and is frequently surrounded by a halo of annular light reflex. It is devoid of blood vessels. At the centre of the macular region is a small depression, the fovea, which is lighter in colour and often glistens. Pathological changes in the macular region are important, as they produce a greater reduction of vision than similar changes in any other part of the fundus.

The periphery of the fundus. This area can be examined only if the pupil is dilated with a mydriatic. Certain disease processes start in this region, for example retinal tears and retinitis pigmentosa.

The following is a brief description of the chief changes met with in the fundus which are important medically.

Papilloedema (Plate XIII). This is a passive oedema of the optic nerve-head, most commonly due to raised intracranial pressure. There is an absence of inflammatory changes, and frequently there is little or no disturbance of visual function. In the initial stages of the condition there is an increased redness of the disc with blurring of its margins, the blurring appearing first at the upper and lower margins, particularly in the upper nasal quadrant. The physiological cup becomes filled in and disappears and the retinal veins are slightly distended. Spontaneous pulsation of the retinal veins is absent.

As the condition progresses the disc becomes definitely swollen. In order to measure the degree of swelling of the disc, it is necessary to start with a high plus lens in the ophthalmoscope and reduce the power of the lens until the centre of the disc is just in focus. The retina, a short distance from the disc, is then brought into focus by further reduction of the power of the lens. This further reduction indicates the degree of swelling of the disc (3 dioptries is equivalent to 1 mm of swelling).

If papilloedema develops rapidly, there will be marked engorgement of the retinal veins with haemorrhages and exudates on and around the disc, but with papilloedema of slow onset there may be little or no vascular change, even though the disc may become very swollen. The retinal vessels will, however, bend sharply as they dip down from the swollen disc to the surrounding retina. The oedema may extend to the adjacent retina, producing greyish-white striations near the disc, and a white macular fan between the fovea and disc may develop in some cases.

Papilloedema occurs in almost 80% of all cases of brain tumour, but is particularly liable to occur in children with tumours of the cerebellum and fourth ventricle. It is uncommon in patients with pituitary tumours. An acute form of papilloedema with haemorrhage extending into the vitreous is characteristic of subarachnoid haemorrhage. A subdural haematoma may produce a similar clinical picture to that of a cerebral tumour. Papilloedema is uncommon in acute meningitis, but is more common in subacute and chronic meningitis. It may be the only physical sign in benign intracranial hypertension. Papilloedema occurring in malignant hypertension is accompanied by arterial changes characteristic of this condition and the haemorrhages and exudates extend far beyond the region of the disc.

Optic neuritis. Inflammatory, demyelinating, or vascular disease may attack any part of the optic nerve, producing an optic neuritis, the characteristic symptom of which is loss of vision, presenting as either a central scotoma or complete blindness. There is often pain on moving the eye, and the pupil on the affected side shows an ill-sustained con-

traction to a bright light. It is customary to divide optic neuritis into papillitis and retrobulbar neuritis.

Papillitis. This is present when the disease process affects the optic nerve head, producing hyperaemia and some swelling of the optic disc. It must not be confused with papilloedema, in spite of their similar ophthalmoscopic appearances. The two conditions can usually be distinguished by the gross visual loss that occurs with papillitis, as compared with the often minimal loss in papilloedema. In papillitis the swelling of the optic disc is usually slight, the distension of the retinal veins is less marked than in papilloedema, and there may be signs of inflammation (hazy vitreous, retinal exudates).

Retrobulbar neuritis. This is present when the disease process affects that part of the optic nerve behind the eye. The same severe visual loss occurs as in papillitis, but the optic disc appears normal in the acute stage of the disease. Both papillitis and retrobulbar neuritis may be followed by optic atrophy.

Optic atrophy. In this condition the optic disc is paler than normal and may even be white (Plate XIV). Because of the wide variation in colour of the normal disc, a useful sign of optic atrophy is the reduction in capillaries on the disc. In optic atrophy the number of capillaries that cross the disc margin is reduced from the normal of 10 to 7 or less. From the appearance of the disc it is customary, although not always very useful, to divide optic atrophy into primary and secondary types. In the *primary* type the disc is flat and white with clear-cut edges. *Secondary* optic atrophy follows swelling of the optic disc, due either to papilloedema or to papillitis. The disc is greyish-white in colour; it may be slightly swollen and its edges are indistinct.

Optic atrophy may occur in a number of disorders, of which the following are a few.

1. Interference with the blood supply to the optic nerve, as in occlusion of the central artery of the retina.

2. Pressure on the nerve, whether in its intra-ocular, intra-orbital, intracanalicular or intracranial portions.

3. Following optic neuritis.

4. Following trauma where the optic nerve or its blood supply is involved.

5. In toxic conditions due to substances such as tobacco, alcohol, lead, etc.

6. In certain congenital disorders, when it is frequently associated with other neurological signs.

7. Following widespread chorioretinal inflammation or degeneration.

Opaque or medullated nerve fibres. These usually present as one or more bright white patches radiating for a short distance from the optic disc (Plate XIII). The patch has a characteristic feathered edge and retinal vessels may disappear for a short distance within it. This condition is a harmless and stationary congenital anomaly.

A myopic crescent. This is a ring of exposed white sclera, usually on the temporal side of the optic disc (Plate XIII), but in some cases extending all round it. When marked it may be associated with other degenerative changes in the fundus, which, if they involve the macula, will result in reduction of central vision.

Retinal haemorrhages. These occur in a number of different conditions and are due to one or more of the following factors.

1. Increased blood pressure within the retinal vessels, as in hypertension and chronic nephritis.
2. Abnormalities in the walls of the retinal vessels, as in arteriosclerosis, diabetes mellitus and occlusion of the central vein of the retina.
3. Abnormalities in the circulating blood, as in severe anaemia, leukaemias, and bleeding diatheses.
4. Sudden reduction in intra-ocular pressure, following a penetrating wound (surgical or traumatic) of the eye.

When superficial, within the nerve fibre layer of the retina, the haemorrhages are elongated and 'flame-shaped', whereas when deep they are round blotches or spots. Subhyaloid haemorrhages, situated in front of the retina, are occasionally seen as very large round haemorrhages with a straight horizontal upper border; they sometimes occur in diabetic retinopathy and after a subarachnoid haemorrhage.

Retinal arteriosclerosis. This occurs either as an exaggeration of the general ageing process of the body or in association with hypertension. It is characterized by (a) broadening of the arterial light reflex, producing a 'copper wire' or 'silver wire' appearance; (b) tortuosity of the vessels; (c) nipping, indentation or deflection of the veins where they are crossed by the arteries; (d) white plaques on the arteries; and (e) 'flame-shaped' haemorrhages and 'cotton-wool' exudates in the region of the macula.

Hypertensive retinopathy (Plate XIV). This is characterized by a generalized narrowing of the retinal arteries, particularly in the young patient. In older patients these changes are masked by the accompanying arteriosclerosis. If the hypertension is severe, fullness of the retinal veins and 'flame-shaped' haemorrhages occur around the optic disc, and

there is retinal oedema extending towards the macula, sometimes accompanied by a star-shaped collection of white exudates around the macula. In malignant hypertension papilloedema is also present. The retinopathy seen in some cases of acute and chronic nephritis is due to the associated hypertension.

Diabetic retinopathy (Plate XIV). The fundamental change in this condition is the formation of capillary micro-aneurysms, seen as tiny red spots around the macula. Micro-aneurysms are not seen in such abundance in any other condition. Retinal haemorrhages and exudates may occur; the haemorrhages are punctate or round, and the exudates have a waxy yellow-white appearance. The haemorrhages may extend into the vitreous and result in a glial proliferation called retinitis proliferans, which may result in blindness by covering the macula or by causing a retinal detachment. Patients with diabetic retinopathy often have associated arteriosclerotic or hypertensive changes in their fundi.

Retinopathies in disorders of the haemopoietic system. In severe anaemias the fundus may be paler than normal and a few small 'flame-shaped' haemorrhages and small woolly exudates may be present.

In polycythaemia the retinal vessels are dark, tortuous and dilated. There may be oedema of the optic disc, and a few retinal haemorrhages may be observed.

In the leukaemias the retinal veins may be tortuous and dilated. In later stages of these diseases the arteries and veins may be yellowish in colour, and the fundus may have a generalized pallor. Retinal haemorrhages of various types may occur, the characteristic ones in leukaemia being round with a pale centre.

Occlusion of the central artery of the retina. The optic disc and surrounding retina are pale, and there is a cherry-red spot at the macula which contrasts with the milky pallor of the adjacent retina. The retinal arteries are narrow or even thread-like.

Occlusion of the central vein of the retina. There is intense swelling of the optic disc, with gross venous dilatation, and numerous retinal haemorrhages extend from the disc in all directions.

Choroiditis. In acute choroiditis there are one or more round or oval whitish patches in the fundus, lying deep to the retinal vessels. These patches have ill defined edges and the vitreous may be hazy. When the acute phase subsides, flat white scars with pigment around their edges are left. The numerous causes of choroiditis include tuberculosis, syphilis (which may cause a disseminated choroiditis), and toxoplasmosis (which characteristically produces lesions at the maculae).

THE EAR

Hearing

Hereditary deafness, usually recessive but sometimes dominant or sex-linked, is commonly the cause of hearing defects dating from birth. Many syndromes are described. Damage to hearing may occur from the effects of viruses on the acoustic apparatus of the fetus by maternal rubella, from teratogenic drugs, from prolonged anoxia or prematurity or in any form of severe jaundice in the newborn. The mother should be warned if her baby is 'at risk', and she can help by noting its reaction to sounds in its environment. The baby can be conditioned to interesting sounds (like the ringing of a tiny bell) just before feeds, and its responses noted if the feed is delayed. Unless the baby is able to hear the sounds of language, its neural mechanisms of speech will not develop normally.

Routine group audiometry tests are regularly conducted by school medical officers. If any abnormality is discovered, the external ear passage and the tympanic membrane of the child should be examined with an auriscope, in case there is a simple cause for the deafness (ear wax, foreign body, infection, unsuspected congenital abnormality). It must be remembered that a child at school may lapse into inattention or become discouraged and irritable, if it has to 'listen' actively under strain for long periods, instead of hearing easily and naturally. A child may be partially deaf in only one ear and have difficulty in focusing on sound, and yet his problem may escape casual notice.

The hearing of adults is often poor because of middle-ear infections in early childhood, which cause scarring, chronic infection or residual perforation; virus infections damaging the cochlea; trauma from head injuries, excessive exposure to noise or sudden changes in air pressure (barotrauma); tumours; simple collections of ear wax; otosclerosis, which produces a conduction deafness due to extra bone having been laid down round the oval window of the middle ear; late syphilis; presbycusis; or the hearing loss may simply be due to an upper respiratory infection with auditory (eustachian) tube obstruction.

The effect on hearing of noise in industry is becoming of more and more importance. Any background noise that necessitates raising the voice during normal conversation is said to be 'traumatic to hearing'. Continuous exposure to noise tends to produce permanent damage to perception of hearing, whereas intermittent exposure often allows recovery to take place. Permanent hearing loss from exposure to noise usually begins with cochlear damage at about 4000 Hz (high-tone deafness) and spreads to tones above and below that point.

Tests of hearing will be found on p. 232.

Audiometry

The audiometer is a simple instrument which allows the examiner to select on a dial a series of pure tones for testing air and bone conduction. Another dial allows the examiner to increase or decrease the intensity of each tone. The patient wears earphones, connected with the instrument, and indicates to the examiner when he begins to hear each separate tone. A graph or audiogram is then constructed which shows how much sound of different frequencies must be intensified to be heard by the patient (Fig. 51). A series of such audiograms gives a good indication of the progress of the disease. The audiogram illustrates clearly the difference between conductive deafness (bone conduction better than air conduction) and sensorineural deafness (air conduction better than bone conduction). There are many specialized tests which differentiate sensory (cochlear) from neural (nerve) deafness.

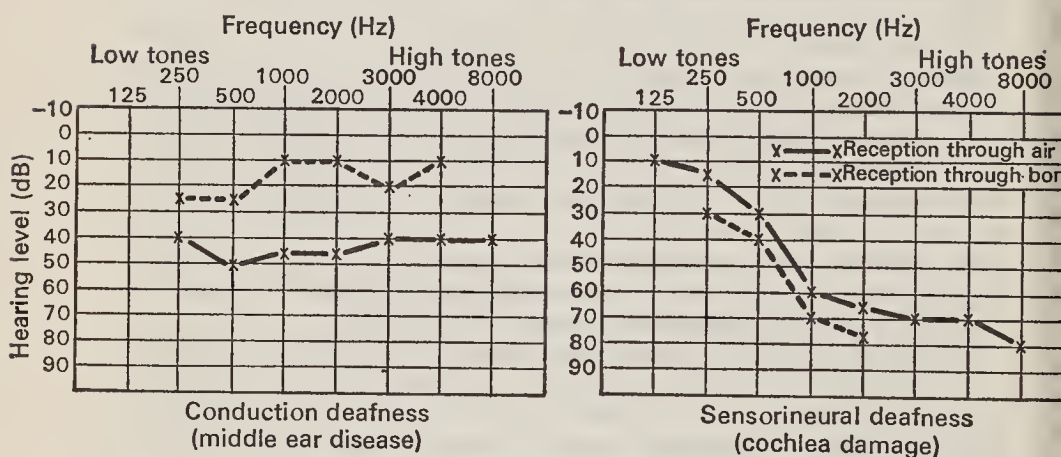


Fig. 51. Audiograms. In conductive deafness bone-conducted hearing is better than air-conducted, and low and high tones are equally affected. In sensorineural deafness air-conducted hearing is better than bone-conducted, and high tones are particularly affected, but different patterns of pure tone loss may be seen. 0 = perfect hearing.

Tests of vestibular function

Disturbance of labyrinthine function, with intermittent attacks of rotational vertigo (objects rotating round the patient), nausea or vomiting and nystagmus, are fairly common and, because the vestibular apparatus and the cochlea share the same circulation, there is often associated deafness and tinnitus in the affected ear. These disturbances occur in Ménière's disease; in late syphilis; after head injuries, when the

inner ear or vestibular pathways have been affected; when tumours in the posterior fossa exert pressure on the vestibular nerve; in vestibular (viral) neuronitis; and when the blood circulation is interfered with in vertebral or basilar artery insufficiency.

The most satisfactory tests for vestibular function are caloric tests, because they are simple and inexpensive and the results are remarkably constant.

The patient lies supine on a couch, with his head raised 30° , to bring the lateral semicircular canal into a vertical plane so that it responds best to thermal stimulation. A stimulus of cold water 7°C below body temperature (i.e. 30°C) and then, after an interval, one of warm water 7°C above body temperature (i.e. 44°C) is applied directly to the tympanic membrane and so indirectly to each labyrinth. Not less than 250 ml water is run into each external ear passage for 40 seconds continuously. In the normal person this produces horizontal nystagmus

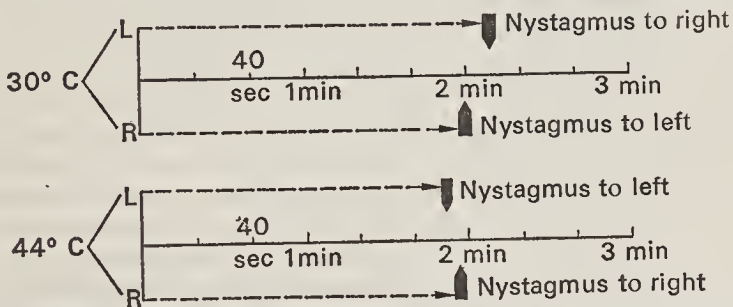


Fig. 52. Caloric tests. Normal average response to stimuli applied separately to each tympanic membrane. The duration of after-nystagmus from the beginning of the stimulus is shown by the dotted line. The direction of the after-nystagmus in terms of the quick component is also shown.

lasting approximately 2 minutes (Fig. 52). The caloric test is of value in the diagnosis of organic lesions at all levels of the vestibular system. In peripheral lesions, the commonest abnormality found is canal paresis, and this is frequently demonstrated in Ménière's syndrome, vestibular neuronitis, and acoustic neuroma.

Examination of the ear

Note any unusual appearance of the external ear, and compare one ear with the other. Though there are many small variations in the shape of the 'normal' ear, occasionally genuine malformations occur, sometimes recurring in different generations of the same family, and sometimes associated with other deformities elsewhere, e.g. asymmetry of the face, cleft palate, spina bifida.

The commonest abnormality met with is probably the protruding ear or 'bat's ear'. Small accessory cartilaginous auricles may be detected, or a tiny fistula, because the development of the external ear is closely related to the branchial cleft between the first and second arches of the developing embryo. There may be only a rudimentary pinna, sometimes combined with the absence of any external auditory passage and this, of course, prevents normal hearing.

Movement of the pinna of the normal ear does not give rise to discomfort, nor does lying on it. It is best to ask the patient, or the infant's mother, whether the ear is painful to the touch, before the examination begins. Always examine the 'good ear' first. Note any swelling or redness that might indicate infection in the skin (such as a small hidden boil), and note any bruising or cut in the skin due to recent trauma. Look at the opening of the external ear passage by gently drawing the pinna towards you with your finger and thumb. Note any desquamation or fissuring, or dermatitis aggravated by scratching with the finger, and glance at the hair and scalp for other evidence of dermatitis.

Make a careful note of any discharge from the opening of the ear passage. Thick pus may be coming from a recently ruptured boil; mucopus could be escaping from a perforation in the tympanic membrane, because the middle ear is lined by mucus-secreting membrane; blood or cerebrospinal fluid may be escaping from a tear in the tympanic membrane from a fractured base of the skull, or blood alone may be coming from any recent tear in the skin of the ear passage.

It is often helpful to determine whether ear pain or ear discharge is of superficial origin or involves the middle ear. Middle-ear infections are nearly always secondary extensions of some pre-existing pharyngeal infection and the infection reaches the middle ear via the Eustachian tube, so that the history of any recent upper respiratory infection is a help in diagnosis. The external cartilaginous ear may be moved freely without adding to the patient's discomfort. If, on the other hand, the ear tenderness is due to a dermatitis, boil or cut in the external cartilaginous ear passage, any movement of the external ear will increase the patient's discomfort. Hearing is unaltered if the lesion is only a small boil or superficial cut, unless swelling of the skin itself occludes the air space of the lumen of the external ear passage. Hearing is always altered in middle-ear infections or haemorrhage. Of course, it is possible to have a middle-ear infection and a dermatitis of the external ear passage at the same time, but more often the site of the lesion can be quite clearly defined.

Acute mastoiditis is not a common complication of ear infections today, thanks to early recognition and treatment of middle-ear infections with efficient antibiotics. Mastoiditis, which is a spread of infection from the middle ear cleft of the child to the mastoid, is an osteitis or

osteomyelitis of the mastoid process. Pain or tenderness from mastoiditis is deep-seated, subperiosteal or periosteal whereas the pain from a boil or from cellulitis tends to be superficial.

The auriscope

To examine the ear further, it is necessary to see the tympanic membrane. This must be illuminated because the external auditory passage is only about 8 mm wide in its greatest diameter and is about 3 cm long. It is best to use a hand auriscope, powered by a small electric battery, which provides adequate magnification, so that early changes in the tympanic membrane, indicating inflammation, may be recognized.

A speculum of suitable size is fitted to the auriscope and the end of the speculum is then gently placed at the opening of the external ear passage, and the examiner looks upwards and forwards to view the tympanic membrane. For the right ear, the examiner holds the shaft of the auriscope in his right hand, like the shaft of a pen; with his other hand he gently pulls the pinna towards him, to straighten out the cartilaginous part of the passage. For the left ear, he holds the auriscope in his left hand. There are advantages in holding the shaft of the auriscope horizontally; for then, if the patient's shoulder is raised unexpectedly in defence, it will not 'jog' the instrument. The little fingers (of both hands) can rest on the patient's skull in front of or behind the ear under examination, and so prevent hurt to the patient should a child make any sudden or unexpected movement of its head.

The normal external auditory passage is very sensitive and is protected at its opening by hairs, sebaceous glands, and apocrine ceruminous glands, but there are no hair or glands in the skin of the inner two-thirds, and here the skin lies directly upon the periosteum of the bone. The examiner should first practise a little with his auriscope on the skin inside his own partly closed fist, and he will rapidly learn how close he should place his own eye to the lens of the auriscope and how to adjust his own accommodation to the magnification. The focal length of the auriscope's lens is already fixed by most instrument makers and requires no adjustment, and the degree of magnification is standard for all general-purpose auriscopes. A gentle examiner will see more than one who is rough and inconsiderate, and an ear should be treated with as much respect as an eye.

Sometimes there is difficulty in seeing a tympanic membrane clearly. The commonest causes of difficulty are:

1. A fading electric battery, giving a poor light.
2. Collected wax or cerumen, obscuring the view.
3. A dirty lens on the auriscope.
4. Faulty setting of the light bulb, so that the beam of light is not properly directed through the aperture of the speculum.

5. In newborn infants, the tympanic membrane is lying almost horizontally, and it will only become elevated to 55° as the child grows older; failure to remember this can cause difficulty in the small infant.

The tympanic membrane

The tympanic membrane (Fig. 53) is oval shaped, and is covered by a thin extension of skin from the walls of the external ear passage. This skin covers the fibrous layer of the membrane, which, on its inner sur-

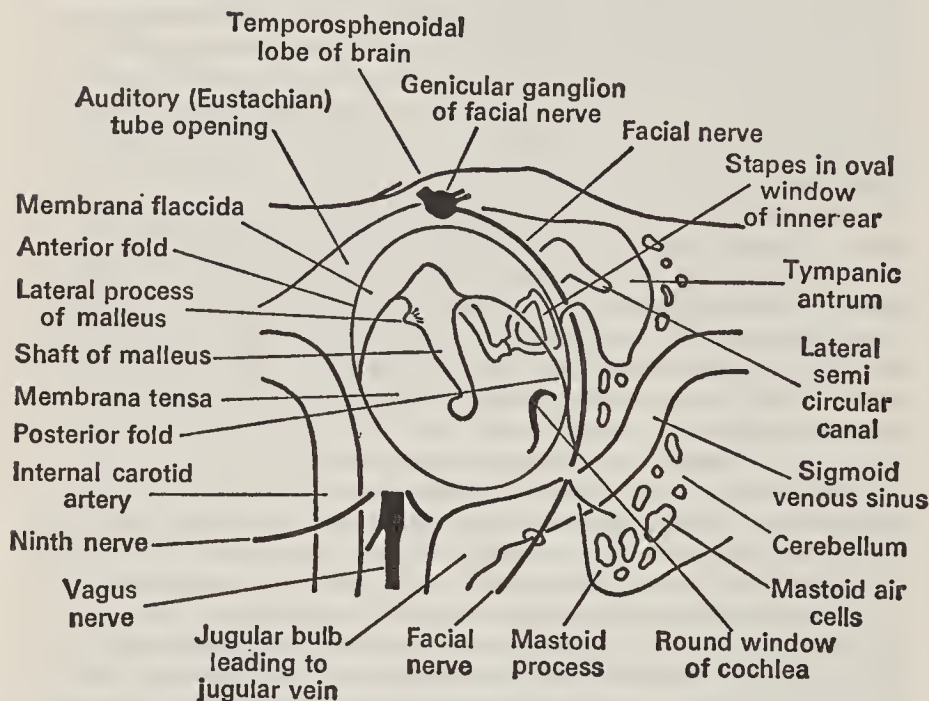


Fig. 53. The tympanic membrane and important anatomical structures around the middle ear.

face, is lined by the moist mucous membrane of the middle ear. The lower three quarters of the membrane appear tense and have a faintly bluish tinge of colour, because of the shadow cast by the middle ear cavity behind it. The upper quarter of the tympanic membrane is pink in appearance and is more flaccid than the lower part. Near the centre is the shaft of the malleus, which is of a pale cream colour, and it is convenient for the examiner to identify the malleus first, in order to orientate himself. Sometimes necrotic middle-ear disease may have destroyed the shaft, and then the examiner should identify the lateral

process, which often survives. The entire tympanic membrane may have been destroyed by disease, and the examiner will then identify some of the anatomical structures of the exposed middle ear (Fig. 53). After recurrent middle-ear infection white plaques of tympanosclerosis are often seen within the structure of the healed drum.

Any unusual redness or bulging of the tympanic membrane should be noted. Bulging or convexity of the tympanic membrane may indicate fluid, pus or increased air pressure in the middle ear and retraction or concavity often indicate that the eustachian tube is obstructed. If the auditory tube remains closed for long periods, serous fluid may collect in the middle ear, and then this may be seen through the tympanic membrane as a fluid level which moves and forms bubbles which are easily distinguishable. Chronic Eustachian tube obstruction eventually leads to retraction pockets of the drum and to secondary fixation of the malleus and incus.

If a perforation of the tympanic membrane is present, it should be described according to its anatomical position. There may be evidence of a recent tear in the tympanic membrane, with fresh or dried blood, after a head injury. It is always important to examine with an auriscope the ears of any patient, conscious or unconscious, who has recently suffered a head injury, and to make a note of what is seen. Cerebrospinal fluid may be escaping through the ear from the meninges of the brain. Fluid pus, escaping from a perforation, may appear to pulsate, from arterial pulsation transmitted from the carotid (Fig. 53). The perforation may be old, resulting from failure to heal after some forgotten infection. Granulation tissue or a polyp protruding through a perforation is a frequent finding in chronic otitis media and it may lead to a blood-stained discharge. In otosclerosis the tympanic membrane is normal, conductive deafness being due to fixation of the stapes. Osteomas are fairly frequently seen close to the tympanic membrane. They appear as rounded hard swellings and are common in swimmers. A cholesteatoma is often associated with chronic middle-ear disease. It consists of layer upon layer of desquamated epithelium. It has a pearly-white appearance and is important, because it may not only destroy hearing but lead to intracranial complications. Glomus body tumours are rare, but appear as reddish swellings in the floor of the middle ear and are associated with paralysis of the ninth, tenth, and eleventh cranial nerves. Squamous carcinoma of the middle ear is also rare and is usually preceded by many years of chronic suppuration. It is often pain or facial paralysis that causes the patient to seek advice.

Radiological examinations

Evidence of bone disease, such as bone erosion or sclerosis, may be detected. A fracture may be clearly seen, but may be present but invisible by X-rays, because other bony structures may obscure it. Widening of the internal auditory meati may help in the diagnosis of acoustic nerve neuroma or posterior fossa tumours.

Syringing the ear

Syringing is used to remove obstructions from the external ear passage, mainly collections of cerumen, sebum and keratin (collectively called wax), foreign bodies or pus. The method is to direct a stream of water, at body temperature, along the sides of the ear passage so that it is deflected by the tympanic membrane and forces the obstruction out. There is an advantage in that the water may gradually soften the wax and make removal easier. If removal of wax by this method proves difficult, it should not be persisted with. It is sensible to ask the patient to soften the wax beforehand with olive oil or almond oil, dropped into the ear passage twice daily for a week. Syringing with force is always liable to cause pain and damage. Even gentle syringing should never be used if a perforation in the tympanic membrane is suspected, and never if there has been recent ear pain or evidence of a recent upper respiratory infection, because an inflamed ear drum, softened by oedema, is very easily damaged. No ear should ever be syringed blindly; repeated inspection with an auriscope is essential. The use of suction is now replacing syringing; but this method also requires gentleness and a good light.

Cleaning the ear passage

Thin twists of cottonwool (the twist must be of less diameter than the ear passage itself, to prevent a piston action), are wound on to the end of a thin disposable wooden applicator, so that the twist of cotton extends beyond the applicator by about 6 mm. The twists may be dipped in olive or almond oil to give added comfort, because the ear passage is very sensitive. The twists are gently introduced into the meatus under direct vision with a good light and are gently rotated to remove pus or particles of desquamated skin. Contaminated swabs should be immediately dropped under disinfectant fluid to prevent cross-infection. Repeated inspection with an auriscope is essential.

Children sometimes put foreign bodies in their ear passages. These almost always require removal under a general anaesthetic. Even an apparently simple and readily accessible foreign body becomes impacted very easily and can cause great pain and damage.

Stimulation of the peripheral branches of the vagus in the skin of the external ear passage may cause not only severe pain but laryngeal irritation, vomiting in children, and fainting in elderly people.

THE LARYNX

Examination of the larynx gives information about the adequacy of the respiratory airway and the causes of changes in the voice.

To perform laryngoscopy it is necessary to use a laryngeal mirror. This is a rounded mirror set at an angle at one end of a long thin metal handle (Fig. 54). The idea is to hold the back of the mirror lightly but firmly against the soft palate so that an image of the vocal cords will appear in the mirror.

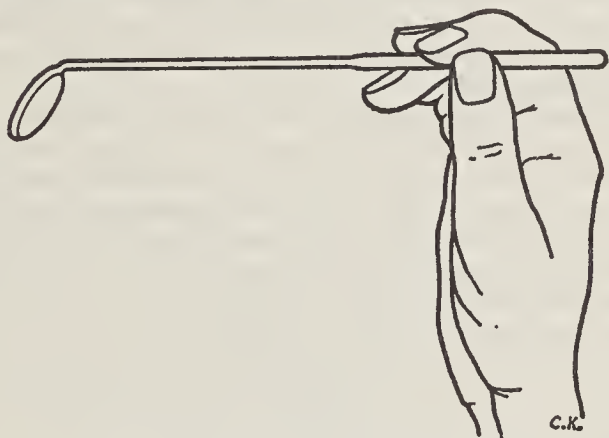


Fig. 54. Holding the laryngeal mirror.

The examiner seats himself facing the patient. He will have to sit very close to his patient, with his legs to the side nearest to the source of light. A standard lamp with a good bright beam is placed at the side of the patient, on a level with the patient's mouth and its beam is directed at the examiner. For a right-handed examiner the light is placed at the patient's left. The examiner may wear a face mask to protect himself should the patient cough directly at him during the examination; most patients with serious lesions of the larynx are very helpful and cooperative, but anxious patients with slight diffuse inflammation of the larynx may be very sensitive and cough easily. Next, the examiner places a head mirror on his forehead, over his right eye. A head mirror is concave and has a hole in the centre through which the examiner's right eye will inspect the image in the laryngeal mirror. The mirror is fastened to the head by a band, and can be adjusted easily because it is fitted

with a ball-and-socket joint. The beam of light from the standard lamp is reflected on to this concave head mirror, then on to the laryngeal mirror, and so down to the larynx. The examiner's eye looks directly down the centre of this reflected light beam, and so the lighted image in the laryngeal mirror becomes clearly visible.

First the patient's mouth is inspected and any dentures are removed. The laryngeal mirror is warmed by placing it in hot water, or holding it for a second over a spirit flame. This to prevent misting of the mirror when the patient exhales. The mirror is wiped clean and tested to make sure it is not too hot by holding it firmly against the sensitive ventral surface of the examiner's wrist.

The patient is asked to lean a little forward, and to open his mouth and put out his tongue. With a small square of clean gauze in the left hand, the examiner takes hold of the tip of the patient's tongue between his thumb and second finger. The gauze square is necessary, or the fingers will slip on the moist tongue. The index finger of the same hand gently lifts the upper lip.

The light from the head mirror is directed at the patient's mouth. The laryngeal mirror is held in the right hand (Fig. 54) and, very carefully in order to avoid touching the tongue, the small round mirror is placed deliberately and steadily against the soft palate near the base of the uvula. The shaft of light from the examiner's head mirror should now shine directly on to the laryngeal mirror, and the laryngeal mirror is gently adjusted until the vocal cords come into view.

Laryngoscopy requires no local analgesic in the great majority of examinations, but it does require the confidence of the patient in the examiner and the examiner's confidence in himself. The examiner must train himself to make rapid and accurate observations.

To facilitate matters, the patient is asked to continue breathing throughout the examination. If the patient is concentrating on breathing he will not think of retching or coughing. If movement of the cords is required, he is asked to say 'ee'.

The *larynx* should be inspected with great care (Fig. 55). The *epiglottis* lies in front, at the base of the tongue. Normally its upper curved edge is clearly seen, its colour is a pale yellow. Any abnormal position should be noted. The base of the *tongue* and the *valleculae* are next examined. Both *piriform fossae* are inspected, and the presence of excessive froth or mucous round the *oesophageal opening* behind the larynx, or in either piriform fossa, should suggest upper oesophageal obstruction or paralysis. In upper oesophageal obstruction, the saliva and mucus from the mouth and upper respiratory tract cannot get away easily, and may overflow into the larynx, explaining the patient's irritating cough.

The *vocal cords* are normally clear-cut, and of a pale-yellow glistening

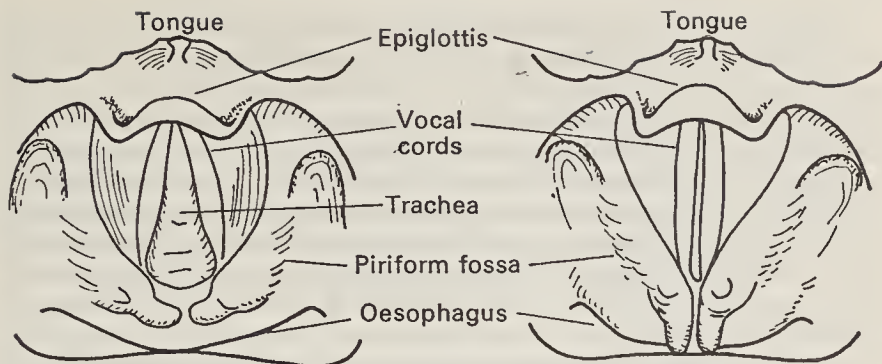


Fig. 55. The larynx as seen in the laryngeal mirror.

appearance. The rest of the laryngeal mucosa is normally a moist pinkish-red colour. Between the vocal cords is seen the lumen of the *trachea*, and the outlines of the tracheal rings. The presence of any pus or mucus in the trachea and any narrowing of the tracheal inlet should be noted.

Displacement of the larynx suggests inflammation or new growth in the tissues closely adjacent to the larynx.

The *epithelium* should be inspected for any irregularity, ulceration, change of colour or presence of oedema. Raised irregular epithelium suggests malignant changes. Ulceration with oedema makes one think of tuberculosis if there is evidence of pulmonary infection. Oedema alone indicates inflammation or trauma, or both. Diffuse redness of sudden onset suggests acute laryngitis.

The *movement* of the larynx should be noted. The movement of both sides of the larynx should be symmetrical. When normal movement is absent, the examiner must ask himself whether some local condition, such as fixation due to malignant changes or myopathy, is preventing normal movement, or whether some lesion along the course of the recurrent laryngeal nerves is responsible. If loss of movement is caused by some local change, there will usually be signs of swelling, or obstruction, or ulceration; or there may be a past history of poliomyelitis or diphtheria.

If the larynx looks quite normal, except for the paralysis, common sites along the courses of the recurrent laryngeal nerves worthy of clinical examination are: (a) the thyroid gland; (b) the apices of the lungs and structures related to the under surface of the aorta; and (c) the cervical oesophagus.

Absence of movement of one or both cords should be carefully noted. If one arytenoid lies a little in front of the other, this is often suggestive of unilateral paralysis and this finding may be helpful if the larynx is difficult to examine.

In *unilateral paralysis* the examiner is struck by the overaction and compensation of the normal cord, which comes right across the middle line to meet the paralysed cord, if the patient endeavours to say the vowel 'ee'. It is this remarkable compensation which explains the apparently normal voice in unilateral paralysis and accounts for the tiredness of the voice towards the end of the day. There is usually no stridor and no apparent obstruction to the airway. Compensation is rapid. Some very temporary difficulty with swallowing is experienced due to unilateral paralysis of the upper sphincter of the oesophagus (cricopharyngeus) which is also supplied by the recurrent laryngeal nerve.

The extraordinary compensatory powers of the larynx are well seen in *bilateral paralysis* where, although the voice is undoubtedly weak, it is remarkably clear, those muscles not supplied by the recurrent nerves taking on added functions. Even when the complete larynx has had to be removed, because of malignant changes, and a permanent tracheotomy is the only respiratory passage, many patients develop an amazingly good voice. Folds in the pharyngeal walls are used for phonation and the relaxed oesophagus makes an efficient air reservoir.

In unilateral paralysis, the patient does not complain of shortness of breath on exertion; whereas in bilateral paralysis there is great narrowing of the laryngeal airway, because the main abductors of the cords are paralysed, and the patient does complain of real shortness of breath on exertion. There is usually acute distress if both cords are paralysed suddenly, but with reassurance, and when adaptation is established, most patients complain of little difficulty in breathing when at rest, or during light movement. The *stridor* during sleep is very inconvenient for others. There is no stridor in unilateral paralysis. It must be remembered that recovery does take place more often than is usually realized, in both unilateral and bilateral paralysis of the recurrent laryngeal nerves.

It is very important to examine the larynx in any case of *hoarseness* that has persisted for 2 or 3 weeks. Intermittent attacks of hoarseness, or loss of voice, with a complete return to normal between attacks, should suggest simple laryngitis or hysteria. If any growth is present on either vocal cord, the voice will never clear until the growth has been removed. Whenever the epithelium of the larynx is ulcerated or irregular, an X-ray of the lung fields, and tests for syphilis should be done before any question of biopsy is considered. If there is purulent sputum, this also should be examined.

Squamous carcinoma arising on the surface of a vocal cord is one of the commoner malignant tumours in men and is not rare in women. In chronic laryngitis there may be polypoid thickening at the anterior end of one or both cords. Nodules due to vocal strain and abuse are usually unilateral. Papillomas are seen in both children and adults. In all such

cases direct laryngoscopy under general anaesthesia is indicated, with biopsy or excision of the lesion. Microlaryngoscopy is often employed today.

No laryngoscopy is complete without an examination of the *neck*. The position of the thyroid cartilage and trachea should be examined and any displacement noted. Movement of the larynx on swallowing should be free and any unusual lump or enlarged nodes must be felt for.

THE NASAL PART OF THE PHARYNX

Examination of the nasal part of the pharynx is carried out in much the same way as that described above for laryngoscopy, and with the same precautions; only here the mirror is smaller, and its handle is bent to avoid touching the tongue. The tongue may be depressed with a spatula, and the small mirror is placed just behind the soft palate facing upwards. The examination requires considerable skill and confidence, and, as with laryngoscopy, some patients are easy to examine, and some very difficult indeed.

The *posterior nares*, lying to either side of the sharply defined vomer, come into view in the mirror, and the posterior ends of the inferior and middle conchae can be seen. To either side lie the openings of the *Eustachian tubes*. The presence of any tumour or pus, or change in the normally pink moist epithelium should be noted, and in children the size of the adenoids should be assessed.

THE NOSE

The outside of the nose must be inspected first. Old scars may indicate past trauma. Recent cuts or bruising should be noted. Redness or swelling may be caused by inflammation. Displacement of the *nasal bones*, to right or left, or depression of the bridge suggests trauma. If the bones are firm and painless, the injury will not have been of recent origin. Collapse of the bony or cartilaginous *bridge* may have followed necrosis. With a finger and thumb on the outside of each nostril, the anterior part of the cartilaginous *septum* can be palpated and any gross displacement or thickening of the septum detected.

The skin round the *nostrils* must be inspected for fissuring or redness. The presence of discharge, whether blood-stained, mucopurulent or watery, must be noted, and whether the discharge is confined to one nostril only or comes from both.

To inspect the *vestibule* of the nose and the nasal passages, a head mirror is needed. There are several advantages in using a concave head mirror for examinations of the nose, nasopharynx and larynx. The examiner can look right down the centre of the beam of light which his

head mirror reflects into these narrow and darkened passages. Both hands of the examiner are free, and his eyes are shaded. Other forms of light such as torches or head lamps or lighted speculae can be tried, but they are not as satisfactory as the perforated head mirror with reflected light.

The vestibule of a small child's nose and the openings of its nasal passages can be inspected by tilting the nose upwards with a finger.

To examine an adult's nose properly a *nasal speculum* is needed. The speculum is held in the left hand (Fig. 56) and the light beam reflected from the head mirror is directed on to the patient's nostril. The speculum is closed, and gently introduced into the nostril. The spring is allowed to open a little, but never to its fullest extent. Gentleness is all important.



Fig. 56. Holding the nasal speculum.

The skin of the vestibule extends inwards for about 1 cm, and it is well supplied with protective hairs, particularly in males. The skin of the nasal vestibule is heir to all the common skin diseases. There is a clear line where the pale skin meets the pink moist mucous membrane of the nasal passages. Medially lies the *nasal septum* which is commonly displaced by trauma. A haematoma of the nasal septum causes a swelling which is soft to the touch, unlike the normal septum, which is firm. Note the presence of any ulceration or perforation of the septum, or the presence of any bleeding point. Laterally, is seen the *inferior nasal concha*. This should be moist and pink and, as its epithelium can become pale and greatly thickened in hay fever and asthma, comparison

should be made with the normal. Above the inferior concha can be seen the anterior part of the *middle concha*. The presence of pus coming from under the middle concha strongly suggests purulent sinusitis; for most of the accessory nasal sinuses open under this concha. Nasal polyps appear as moist greyish swellings occluding the air passages. They are easily movable and painless, unlike the conchae, which are fixed and tender. Hyperaemia of the mucosa and the presence of mucopus in the nasal passages, probably indicates infection. A persisting one-sided blood-stained nasal discharge may indicate the presence of a foreign body or malignancy if present in an older person. The presence of any raised irregular or bleeding epithelium should be noted.

The *nasal passages* form part of the upper respiratory tract and should never be examined without reference to the lower respiratory tract. All air reaching the lungs has to pass through the nasal passages to be filtered, warmed and moistened, and any infection or obstruction in these passages may ultimately have some effect on the health and function of the lungs and bronchi. The presence of nasal obstruction must be noted. In children, mouth breathing due to nasal obstruction gives rise to a change in the pattern of facial expression, which is indicated by the open mouth and sagging of the facial muscles. The lips and mouth are dry.

Severe nasal obstruction may prevent air reaching the olfactory area, and so cause loss of the sense of smell.

THE NASAL SINUSES

To examine the *maxillary sinuses*:

1. Palpate the bony walls of the sinus, giving particular attention to the bone under the eye. Compare the outline on the two sides. Any tenderness, swelling, expansion or depression of bone must be observed. Malignant changes in the epithelium of the maxillary sinus may remain undetected for many months. The palate and alveoli must also be inspected, and palpated from inside the mouth.

2. Examine the nasal passages, to detect any evidence of pus or polyps appearing from any of the normal openings of the sinuses.

3. Transilluminate the sinuses. This is done by placing a strong light in the centre of the hard palate. Any upper denture should be removed. The patient closes the lips round the handle of the transilluminating lamp. The examination should be performed in a dark room. Light from inside the mouth shines through the hollow sinuses, producing a light in the bone under each eye, and a reddish reflection from the retinae.

Comparison between the two sides is made. Pus in the maxillary sinus throws a dark shadow. Patients will differ greatly in the thickness

of their maxillary bones. Some transilluminate clearly, others with difficulty. Transillumination is a useful clinical aid, because if the sinuses are perfectly clear on transillumination, an X-ray may be unnecessary.

If any abnormal shadow is found, an X-ray of the sinuses will give additional information, and will help to confirm the clinical examination.

To transilluminate the *frontal sinuses*, the light is placed under the frontal ridge of the orbit. The *ethmoid* and *sphenoid* sinuses are best examined by X-ray, combined with clinical inspection of the nasal passages.

11

THE LOCOMOTOR SYSTEM

The Bones — The Joints — The Gait — Some Investigations used in Rheumatic Diseases

The locomotor system includes the muscles, bones and joints. The examination of the muscles is most conveniently considered along with that of the nervous system (Chapter 10). There remain for consideration the bones and joints.

THE BONES

In examining the *long bones* of the limbs, look for any alterations in shape or outline, for localized swellings in the bone, for signs of fracture and for evidence of undue tenderness. In osteitis deformans (Paget's disease) the bones are both deformed and enlarged. Alteration in the shape of the bones occurs in rickets. Localized swellings are mostly due to surgical conditions. Spontaneous fractures may occasionally assist in the diagnosis of secondary carcinoma, generalized osteitis fibrosa (hyperparathyroidism), osteogenesis imperfecta or multiple myeloma. Undue tenderness of the bones, apart from surgical conditions, is found in generalized osteitis fibrosa, myelomatosis, occasionally in carcinomatosis of bones, and very rarely in leukaemia.

The *vertebral column and skull* demand special attention. Note the presence of any local projections or angular deformity of the vertebral spines, and state which vertebrae are involved and at what level the projection is most prominent. Landmarks are C7 (vertebra prominens) and the last rib, articulating with the 12th thoracic vertebra. In many cases, however, the last rib cannot be distinctly felt and is therefore rather untrustworthy as a guide.

Note also any curvature of the spinal column as a whole, or of part of it, distinguishing such general curvature from the local projections referred to above.

The curvature may be in an anterior, posterior or lateral direction. Anterior curvature (extension deformity) is termed *lordosis* and is commonest in the lumbar region. General posterior curvature (flexion deformity) is spoken of as *kyphosis*. It occurs most typically in the thoracic region in old persons and must be distinguished from the

localized angular deformity of Pott's disease. Lateral curvature is termed *scoliosis*, and may be towards either the right or the left side. It is always accompanied by a rotation of the bodies of the vertebrae in such a way that the spines come to point towards the concavity of the curve, i.e. the curvature is greater than it appears from inspection of the posterior spinous processes. Kyphosis and scoliosis may often be combined.

Test the movements of the spine (p. 296). Note the exact site of any pain and observe whether it is accompanied by reflex muscle spasm. Painful restriction of movement of the cervical and lumbar spine is an important sign of *cervical and lumbar spondylosis* but may also be found in simple strains of the back or neck. The spine may be fixed in *ankylosing spondylitis*, as may also the costovertebral joints.

Certain well recognized types of abnormal *skull* are met with. In *acromegaly* the supra-orbital ridges and bones of the face, particularly the lower jaw, are enlarged, so that in relation to them the calvarium may appear small. In *achondroplasia* the skull, though of approximately normal size, appears very large in contrast to the generally small stature. In addition, the bridge of the nose is depressed and the nostrils tend to point directly forwards.

In *osteitis deformans*, in addition to the widening and bowing of the long bones, the skull is often greatly enlarged, particularly in its transverse diameter, so that it appears to bulge above the ears.

In *hydrocephalus* the skull tends to assume a globular form. The forehead is overhanging and the eyes are pushed down so that the upper part of the sclerotic is exposed. The lateral aspect of the skull (above the ears) project outwards. If the patient is a child, as is usually the case, the fontanelle is wide and bulging, and often fluctuates very distinctly. The sutures may be opened up, and imperfectly ossified areas (*cranio-tabes*) may be detected in the bones.

In *rickets* the skull tends to be square or oblong and box-shaped. The frontal and parietal bones often show central thickening ('bossing'). The forehead, however, does not overhang, nor are the eyes depressed, and although the fontanelle is usually widely open, it does not bulge as it does in hydrocephalus, nor are the sutures opened up.

In *congenital syphilis* the forehead is vertical, the frontal eminences are often exaggerated, and the bridge of the nose is depressed.

THE JOINTS

These should be examined by inspection and palpation, and by tests for their range of movement. It is important to proceed in a routine manner, e.g. the jaw, cervical spine, shoulder girdle and upper limb, thoracic and lumbar spine, pelvis and lower limb, so that inconspicuous but import-

ant joints like the temporomandibular, sternoclavicular and sacroiliac will not be overlooked. Always compare the corresponding joints on the two sides of the body.

On inspection and palpation look for enlargement or irregularity of the joint; for redness, tenderness and heat; and note whether the overlying skin is dry or moist. Tenderness may be recorded in four grades, depending upon the patient's reaction to firm pressure of the joint between finger and thumb. Grade 1 tenderness: the patient says the joint is tender. Grade 2: the patient winces. Grade 3: the patient winces and withdraws the affected part. Grade 4: the patient will not allow the joint to be touched. Grade 4 tenderness only occurs in gout, rheumatic fever or suppurative arthritis. In gout the skin overlying the affected joint is dry, whereas in suppurative arthritis or rheumatic fever it is moist.

Look also for bony outgrowths, such as Heberden's nodes on the fingers in some cases of osteoarthritis, for rheumatoid nodules, for gouty tophi and for atrophy of muscles in the region of the joint.

If the joint is enlarged, determine whether the enlargement is due to effusion into the joint space, when it normally has a characteristic shape and fluctuation can often be elicited; to thickening of the periarticular tissues, such as occurs in rheumatoid arthritis; to irregular bony thickening by osteophytes such as occurs in osteoarthritis; to enlargement of the ends of the bones, such as occurs in pulmonary osteoarthropathy; or to complete disorganization of the joint with absence of pain sense such as occurs in neuropathic (Charcot's) joints.

If tenderness is present localize it as accurately as possible and determine particularly whether it arises in the joint or in neighbouring structures, e.g. in the supraspinatus tendon rather than in the shoulder joint. Feel the joint with one hand, while it is moved passively with the other. A grating or creaking sensation known as *crepitus* may be felt. This often indicates osteoarthritis, but not invariably so, for crepitus is commonly felt in the shoulder joints of older persons, whereas osteoarthritis of these joints is rare.

In examining joints for the range of movement an estimate of the degree of limitation present based on previous experience or on comparison with the normal side may often be sufficient, but for accurate description the actual range of movement should be measured with a protractor or goniometer. Testing the range of *passive* movement is generally more informative than observing *active* movement; although it is sometimes useful to test the latter as well. The greatest possible gentleness must be exercised, particularly in the case of painful joints. Limitation of movement in a joint may be due to pain, muscle spasm, contracture, inflammation or thickening of the capsules or periarticular structures, effusion into the joint space, bony or cartilaginous over-

growths, bony ankylosis, or to painful conditions quite unconnected with the joint.

In describing the range of movement of joints the scheme shown in the following pages (modified by permission of the authors and publishers, from E. F. Cave & M. R. Sumner (1936) *J. Bone Jt Surg.*, XVIII, 455) will be found useful. All motion should be measured in degrees from a neutral position or zero which must be defined whenever possible.

Spine. Neutral position is normal upright position for patient, but cannot be further defined. Test:

1. Forward.
2. Extension.
3. Lateral bending.

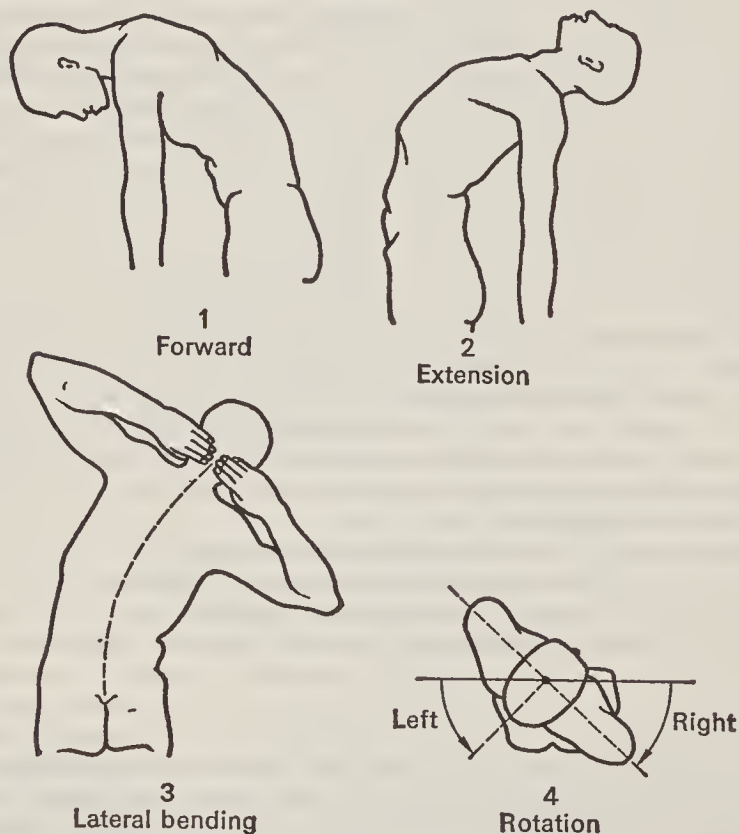


Fig. 57. Movements of the spine.

4. Rotation with pelvis fixed (patient seated) comparing angle made between axis of shoulders and that of the pelvis.

These movements cannot conveniently be measured, but should be compared with the probable normal for the patient's age.

Neck. Neutral position is that with head erect and chin drawn in. Test:

1. Rotation right and left.
2. Flexion.
3. Extension.
4. Lateral bending.

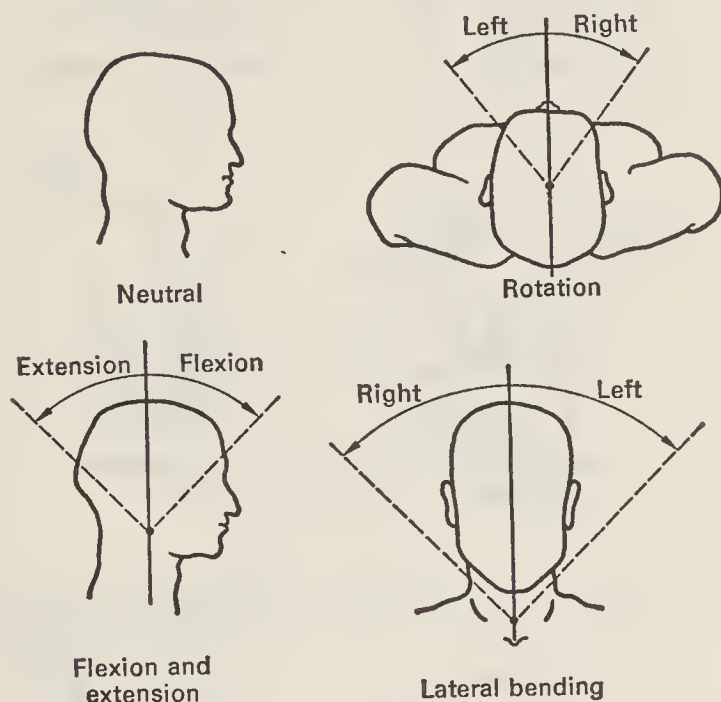


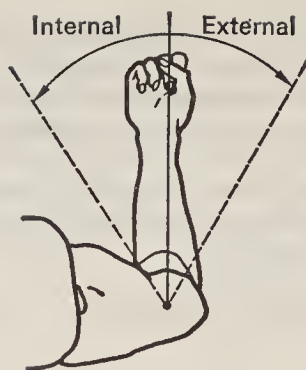
Fig. 58. Movements of the neck.

Shoulder. Neutral position is arm to side, elbow flexed to 90° with forearm pointing forwards. Test:

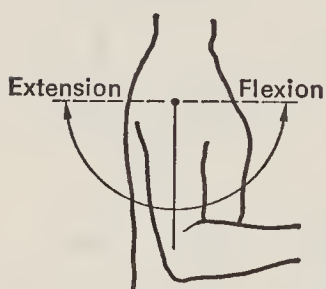
1. Flexion.
2. Extension.
3. Abduction.
4. Rotation in abduction.
5. Rotation in neutral.
6. Elevation (this is shoulder girdle motion as compared with 1-5 which are humeroscapular motion).



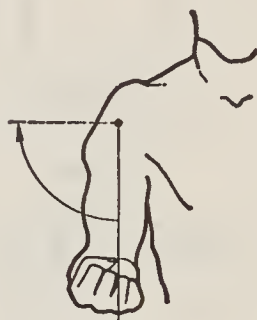
Neutral



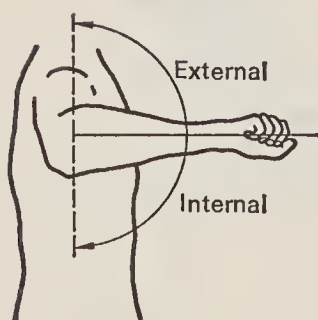
Rotation in neutral



Flexion and
extension



Abduction



Rotation in
abduction



Elevation

Fig. 59. Movements of the shoulder.

Elbow. Neutral position is with forearm in extension. Test:

1. Flexion.
2. Hyperextension.

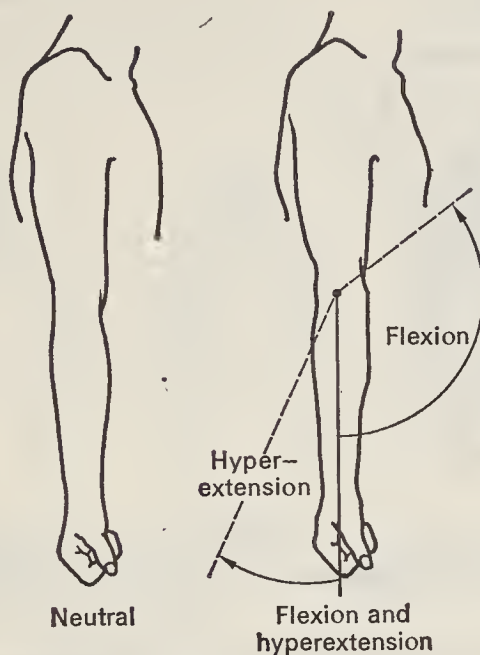


Fig. 60. Movements of the elbow.

Forearm. Neutral position is with arm by side, elbow flexed to 90° , thumb uppermost. Test:

1. Supination.
2. Pronation.

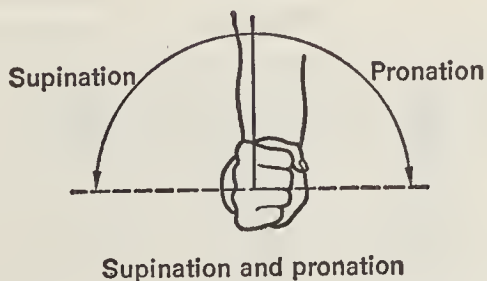


Fig. 61. Movements of the forearm.

Wrist. Neutral position is with hand in line with forearm and palm down. Test:

1. Dorsiflexion (extension).
2. Palmar flexion.
3. Ulnar deviation.
4. Radial deviation.

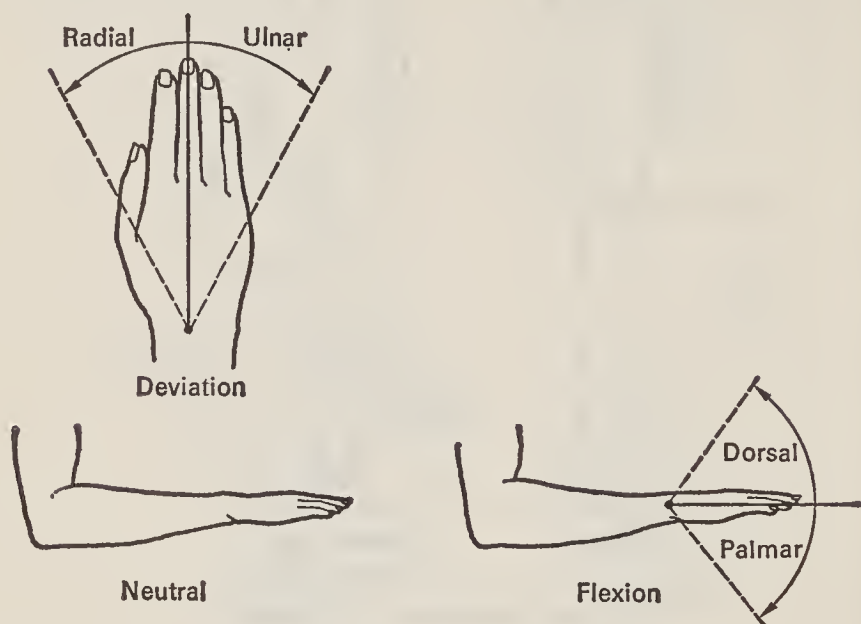


Fig. 62. Movements of the wrist.

Fingers. Neutral position is with fingers in extension. Test:

1. Flexion at metacarpophalangeal and interphalangeal joints.

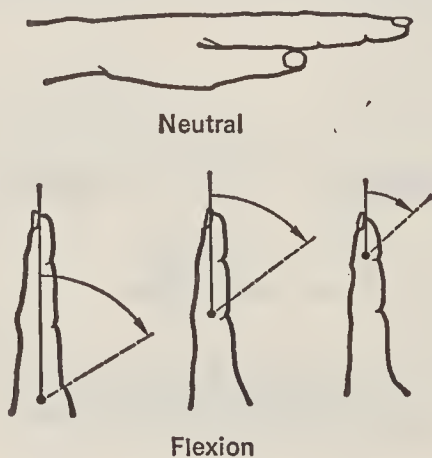


Fig. 63. Movements of the fingers.

Thumb (carpometacarpal joint). Neutral position is with thumb alongside forefinger and extended. Test:

1. Extension.
2. Flexion—measured as for the fingers.
3. Opposition.
4. Abduction (not illustrated) is movement at right angles to plane of palm.

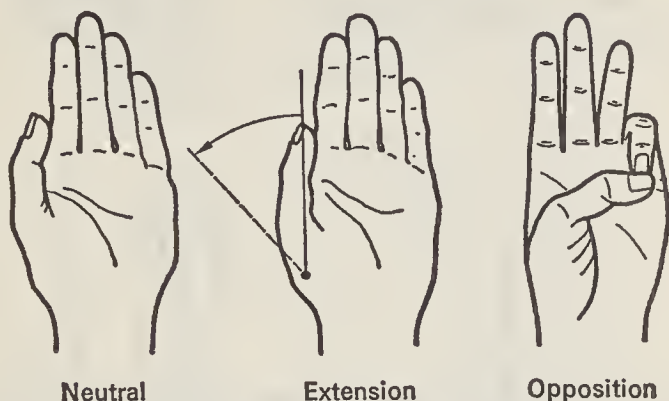


Fig. 64. Movements of the thumb.

Hip. Neutral position is with hip in extension, patella pointing forwards. Test:

1. Flexion measured with knee bent. Opposite thigh must remain in neutral position.
2. Abduction, measured from a line which forms an angle of 90° with a line joining the anterior superior spines.
3. Adduction, measured in the same manner.
4. Rotation in flexion.
5. Rotation in extension.

Additional examinations of the hip joint:

1. Testing for extension. Attempt to extend the hip with the patient lying in the lateral position on the opposite side. Extension should normally be at least 20° from the neutral position. This movement is lost early in hip joint disease and the loss is associated with a flexion deformity and a 'waddling' gait.
2. Measurement of 'true' and 'apparent' shortening. If the length of the legs is measured from the anterior superior iliac spine to the medial malleolus on the same side, any difference is referred to as 'true' shortening, and almost invariably indicates disease of the hip

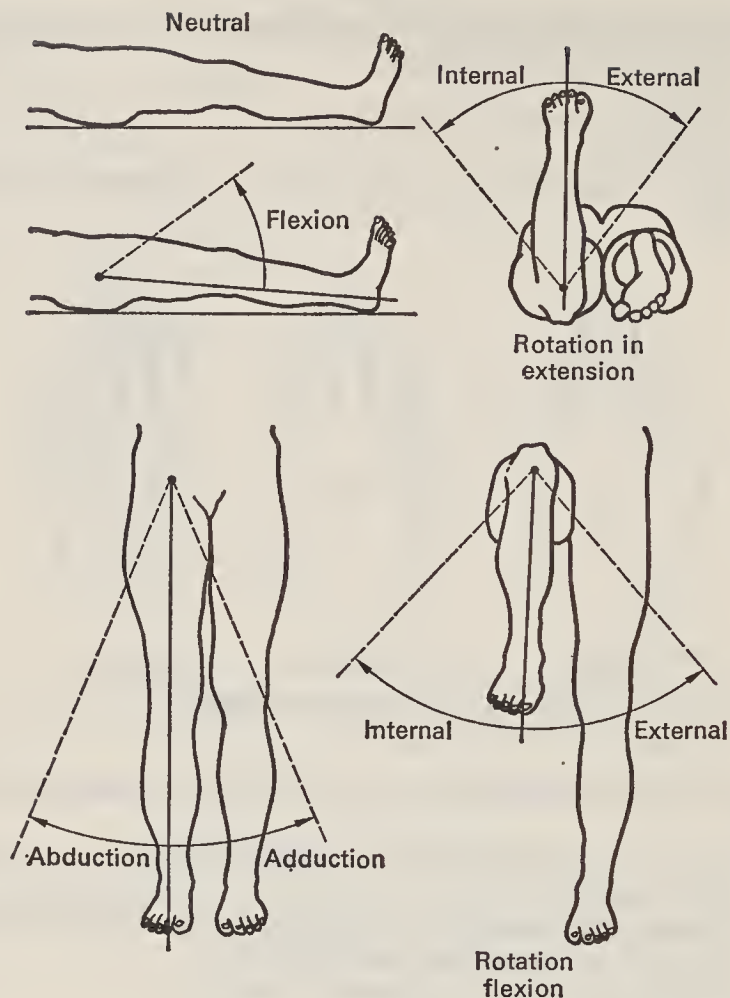


Fig. 65. Movements of the hip.

joint or the neck of the femur on the shorter side. If the length of the legs is measured from the umbilicus to the medial malleoli, any difference is referred to as 'apparent' shortening and may be due either to disease as mentioned above or to tilting of the pelvis, usually due to an adduction deformity of the hip.

Knee. Neutral position is complete extension. Test:

1. Flexion.
2. Hyperextension.

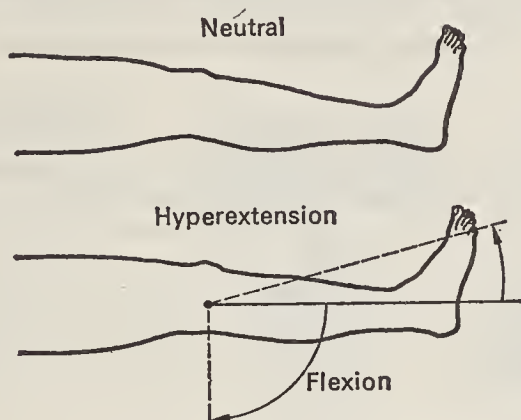


Fig. 66. Movements of the knee.

Ankle. Neutral position is with the outer border of foot at angle of 90° with the leg, and midway between inversion and eversion. Test:

1. Dorsiflexion, with foot in inversion. Test with knee in flexion and extension to exclude tight calf muscles.
2. Plantar flexion.

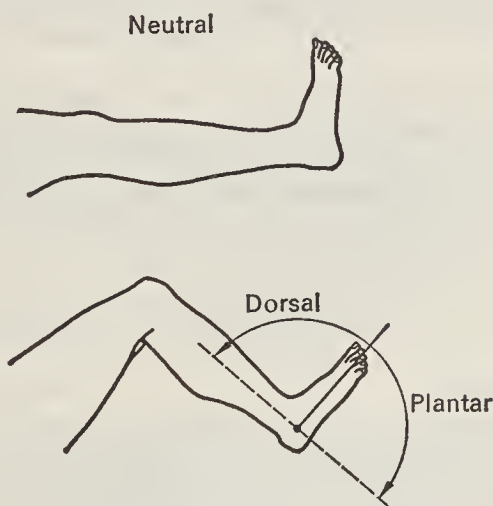


Fig. 67. Movements of the ankle.

Foot. Neutral position cannot be defined. Test:

1. Inversion and eversion at sub-talar joints.
2. Forefoot adduction and abduction at mid-tarsal joints, with os calcis held in neutral position (inversion and eversion may also be tested).
3. Flexion at metatarsophalangeal and interphalangeal joints.

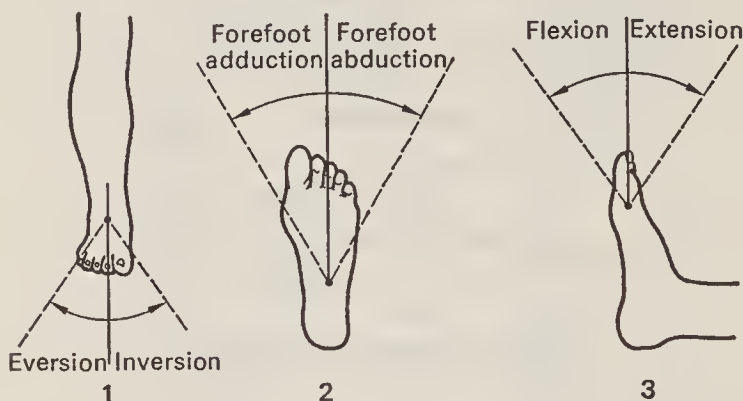


Fig. 68. Movements of the foot.

THE GAIT

When one studies the gait, it is well to have the legs fully exposed. The patient should therefore be unclothed or else wear a small triangle or bathing slip. The feet should be bare. Ask the patient to walk away from you, to turn around at a given point and then to walk towards you again.

Abnormalities of the gait are usually due either to local lesions in the lower limbs or to neurological disorder, though intoxication, hysteria or malingering may occasionally cause difficulty. A full examination of the legs and feet should reveal any local cause, which may range from a painful corn to osteoarthritis of the hip. Abnormalities due to neurological disorder are described in Chapter 10.

SOME INVESTIGATIONS USED IN RHEUMATIC DISEASES

Rheumatoid factors

About 80% of patients with rheumatoid arthritis have in their sera *antiglobulins* (autoantibodies in the IgM, IgG or IgA classes directed against other IgG molecules). These 'rheumatoid factors' may be detected by their ability to clump polystyrene particles coated with

human IgG (*Latex test*) or sheep erythrocytes coated with rabbit IgG (*Rose-Waaler, DAT or SCAT tests*). A significant titre in the latex test is 1/80, in the sheep cell tests 1/32. The former is more sensitive, the latter more specific for rheumatoid arthritis but positive tests may also be obtained in the elderly and in patients with hepatic and other diseases.

Antinuclear antibodies

Autoantibodies directed against antigens present in cell nuclei are highly characteristic of systemic lupus erythematosus. Other systemic connective tissue diseases may give results which are positive, usually less strongly so. Three laboratory tests are most commonly used:

Antinuclear factor test (ANF). An immunofluorescent technique which is highly sensitive, easy to perform and therefore useful as a screening test. The significance increases with the titre. It is always positive in active SLE.

DNA binding test. A radioisotope immunochemical technique detecting antibodies to native (double-stranded) DNA. Expressed as 'percentage binding', results over 30% are positive and the significance increases with higher figures up to 100%. This is the most specific laboratory test for active SLE.

LE cell test. The original test (p. 155) is time-consuming to perform. Peripheral blood 'buffy layer' cells which have been incubated are smeared and stained. Ingestion of nuclear material by leucocytes produces the characteristic 'LE cell'.

Uric acid determinations

A consistently normal plasma uric acid level (below 5.5 mg/100 ml in women and 6.5 mg/100 ml in men) effectively excludes the diagnosis of (untreated) gout. Raised levels occur in a wide variety of circumstances and, in themselves, do not establish the diagnosis of gout. On a low purine diet the 24 hour urinary urate excretion should not exceed 600 mg. Higher levels indicated 'overproducers' of urate who may be at risk of stone formation.

Synovial fluid crystals

Urate and pyrophosphate crystals can be accurately identified in synovial fluids using polarized light microscopy. This provides a positive diagnosis of gout or pyrophosphate arthritis ('pseudogout') respectively. Samples should be submitted without anticoagulant, and sterile.

Antistreptolysin-O (ASO) test

The presence in the serum of this antibody in a titre greater than 1/200 (reported as the reciprocal, i.e. '200') or, more significantly, a rising titre, indicates a recent haemolytic streptococcal infection. A positive test does not establish a diagnosis of rheumatic fever, but a negative test makes this diagnosis unlikely.

12

CLINICAL EXAMINATION OF CHILDREN

General Examination — Special Examinations — Milestones of Development — Examination of the Newborn

The clinical examination of young children is an art which must be learnt by experience. However, since children who object very strongly to being examined usually do so because they are afraid, the beginner can often achieve an adequate examination by patience and a gentle approach. The child and his mother should be greeted in a friendly manner and the child offered a selection of toys suitable to his age. The *history* is then taken from the mother incorporating the special questions set out on p. 16. While talking to the mother, the examiner should observe the patient and take note of certain points. Does the child look well or ill? Is he apathetic or alert, anxious or relaxed? Is there anything unusual about the facies? Are there any obvious physical deformities? Is the child well nourished or wasted? Is there any difficulty in breathing?

GENERAL EXAMINATION

The child should now be at ease and getting used to the strange surroundings. This is the time to record the respiration and pulse rates. The average respiratory rate in a normal newborn child is 40/minute; by the second year it has fallen to 25 or so, and by the fifth year to about 20. A raised respiratory rate in a child at rest usually indicates some disorder of the respiratory or cardiovascular systems. The normal pulse-respiration ratio is 3 or 4 to 1.

The pulse rate is counted in young infants by palpation or auscultation of the chest, in older children by palpation at the wrist. The average pulse rate at birth is about 130/minute, at 1 year 110, at 3 years 100, at 8 years 90, and at 12 years 80. During sleep the pulse rate falls about 10–20 beats/minute. The normal limits for pulse rates are wide. Tachycardia is common and may be due to crying, excitement, exercise and fever as well as to various diseases. Bradycardia is rare and usually indicates some cardiac abnormality. Sinus arrhythmia is present in almost all children, but other irregularities of rhythm are uncommon

even in the presence of cardiac disease. Always feel for the femoral pulses which are absent in coarctation of the aorta.

Examination should now proceed by the usual methods of inspection, palpation, percussion and auscultation; but no set routine can be followed and the examination is by regions rather than by systems. Each patient will, by his reaction to the various procedures, dictate the order of examination and even the position in which he will be examined. Young infants are usually examined on the mother's lap, older children may not be prepared to lie down on a couch and can be examined standing at the mother's side. Most children are prepared to have some of their clothes removed, although they are often modest and should be allowed to keep their pants or knickers on.

It is important to be gentle and to have warm hands. Begin by looking at and feeling the child all over. Observe the general state of development and nutrition. Is the skin dry or moist, and is there the normal degree of elasticity? Is there a rash present? Are there any bruises? Multiple bruises of different ages in children under the age of 3 suggest the possibility of the battered baby syndrome. The shape of the chest and abdomen should be noted. The abdomen is normally rather protuberant in young children. Feel the head and the *anterior fontanelle*: this closes normally between 15 months and 2 years and delayed closure may be due to rickets or hydrocephalus. The posterior fontanelle is always small and closes by the second month. The degree of tension of the anterior fontanelle is important. In health it pulsates and is in the same plane as the rest of the surrounding skull. A depressed fontanelle is a sign of dehydration, and a tense bulging fontanelle indicates raised intracranial pressure. The fontanelle is normally tense when the child is crying.

The *shape of the head* should be noted. It may be abnormally shaped, owing to premature fusion of the sutures. It is globular in hydrocephalus, and is often asymmetrical in normal infants who tend to lie persistently on one side (plagiocephaly).

Inspect the *eyes* for cataracts and conjunctivitis. In infants a squint may be detected by shining a light in front of the face. The two corneal light reflexes are normally symmetrical.

Examine the *limbs*. Look for wasting, swelling and tenderness, and for limitation of movement of joints. A painful limb may be immobile in infants (pseudo-paralysis) or cause a limp in older children. Feel the wrists for widening of the epiphyses of the radius and ulna which is a sign of rickets. Note the presence of deformities such as knock-knee, bow-legs and flat feet.

The *lymphatic glands* should be palpated. Small glands can normally be felt in the anterior and posterior triangles of the neck, the axillae and the inguinal regions. Lymph glands enlarge readily in children as a

result of local conditions such as tonsillitis or generalized diseases such as rubella.

The *abdomen* cannot be palpated satisfactorily in a child who is crying or resisting. Small infants can be given a feed from a bottle to stop them crying, but with an older child one must wait until he settles down before attempting to palpate the abdomen. Children from about 1 to 3 years old will often refuse to be examined lying down, but in this age group the abdomen can be palpated from behind when the child is standing on the mother's lap and looking over her shoulder.

Palpation should be gentle and light. The liver edge can be felt quite easily and in young children normally extends down to 2 cm below the costal margin. The spleen when enlarged can be felt below the left costal margin. Slight enlargement of the spleen is common in children with infections of all types. Faecal masses can be felt in constipated children and a full or distended bladder presents as a firm mass arising out of the pelvis. Abdominal tenderness is best detected by watching the child's facial expression during palpation.

Feel for the testes in the scrotum but remember that an active cremasteric reflex may retract them into the abdomen.

The *thorax* should be inspected for deformity of the chest wall and for intercostal or subcostal indrawing, which indicates respiratory obstruction, chronic lung disease or a cardiac abnormality. Look also for the grunting respirations of the child with pneumonia or other respiratory disease. This is due to a reversal of the normal respiratory rhythm. The grunting expiration is followed by inspiration and then a pause. The thickening of the costochondral junctions ('rickety rosary') which occurs in rickets can be seen and felt. Palpate the anterior chest wall for the cardiac impulse and for thrills. In children under the age of 6 or 7 years the cardiac impulse is normally in the fourth intercostal space, just to the left of the midclavicular line. In older children it is usually in the fifth space in the midclavicular line. Vocal fremitus is not a sign of great value in children.

Percussion of the chest should be light and in small children can be direct, that is to say the chest wall is tapped directly with the percussing finger without the use of a pleximeter finger. The chest is much more resonant in children than in adults.

A stethoscope with a small bell chest-piece is suitable for general auscultation of a child's chest. Listen for the breath sounds and for adventitious sounds. Because of the thin chest wall, breath sounds are louder in children than in adults, and their character is more like the bronchial breathing of adults (puerile breathing). Upper respiratory infections frequently give rise to loud coarse rhonchi, which may be conducted down the trachea and main bronchi.

The normal splitting of first and second heart sounds is easier to hear

in children than in adults. Venous hums and functional systolic murmurs are often heard in normal children (see p. 115).

In older children examination of the *nervous system* can be carried out in the usual manner. In young children the extent of the neurological examination depends on their age and willingness to cooperate. Certain special points should be mentioned. If the child can walk, look for abnormalities of gait and the presence of a limp or ataxia. Note any abnormal movements. Tics or habit spasms are repetitive but purposeful movements, such as turning of the head or shrugging of the shoulders. Choreiform movements are coarse, involuntary, purposeless jerks which follow no particular pattern. These are best demonstrated by asking the child to hold out his arms in front of him. In this position the child with chorea adopts a characteristic posture, with the wrists in flexion and the fingers in hyperextension. Coordination is tested by some modification of the finger-nose test, for example reaching out to touch a toy held in the examiner's hand.

Muscle tone and muscle power should be assessed. The child with marked hypotonia will slip through one's hands when picked up under the armpits. Meningeal irritation or spasm of the spinal muscles is detected more readily by resistance to passive flexion of the head and neck (neck stiffness) than by testing for Kernig's sign.

Tendon reflexes are often difficult to elicit in normal children, and some time may be needed to find the correct position of the limb for this purpose. Only gentle percussion of tendons is required and this is often better done with the examiner's finger than with an adult-sized patellar hammer. The plantar responses are extensor in normal infants up to the age of about 18 months, and the persistence of an extensor response after the age of 2 years indicates an upper motor neurone lesion.

With a little ingenuity, most of the cranial nerves can be tested. For example, by getting a baby to follow a bright object moving in various directions the eye movements can be studied and the presence of nystagmus noted. Examination of the fundi is particularly difficult in infants, as they cannot fix their gaze. Infinite patience is needed for this manoeuvre, as it entails waiting with the ophthalmoscope in position for fleeting glimpses of the optic disc. Forcible attempts to keep the eyes open only make the procedure more difficult. Older children will often fix their eyes on a toy held up behind the examiner by an assistant.

The testing of vision, hearing and certain motor functions in young children is included in the developmental screening examination described on p. 321.

For the child the most unpleasant examinations are those of the *ears, nose, mouth* and *throat* and these should be left until last. With skill, gentleness and patience, even these procedures can usually be carried out without making the child cry. Start with the nose, which need only

be examined superficially. By placing a shiny surface, such as a mirror, under the nostrils, patency of the nasal airways can be judged by the size of the area of clouding. With a good light, the appearance of the mucous membrane of the anterior nares can be inspected. Look for the pale swollen inferior turbinates which are characteristic of allergic rhinitis. Proceed next to examine the external auditory meati and tympanic membranes. Allow the child to see and handle the auriscope and speculum, using the instruments as a toy in a simple game for a few moments. If, in spite of these preparations, the child still resists, he will have to be held by his mother. Sit the child on her lap facing to one or other side, and get her to hold the child firmly with one arm around his head and the other around his upper arms and shoulders. Held in this way, the child can be kept still long enough for the ear drums to be inspected.

The mouth and throat are examined in similar fashion. A cooperative child can be encouraged to 'show his teeth' and thus open his mouth without the use of a spatula, which so many children dread. The uncooperative or very young child must be held by the mother, as for the examination of the ears, but in this case the child sits facing the examiner. The use of a spatula may be unavoidable. Force it gently between the teeth and on to the tongue, which is then depressed. Note the state of the teeth, the tongue, and the mucous membrane of the mouth. Look for Köplik's spots, the pharyngeal lesions of chicken pox, and the white patches of thrush. Inspect the tonsils and the pharynx. Look for streaks of mucopus on the posterior pharyngeal wall (post-nasal drip).

SPECIAL EXAMINATIONS

The following examinations need special mention.

Measurements

Measurements of weight and height are important in the examination of children. Height can be measured only in children over the age of about two years. Below this age supine length can be measured roughly with a tape measure. All measurements should be made under standard conditions, and children should be weighed unclothed. One of the particular features of childhood is that it is a period of growth, the pattern of which may be adversely affected by many disturbances of health. Heights and weights should be compared with those of healthy children of similar sex, age and build, and for this purpose the percentile charts (Figs 69–74) are invaluable. Serial measurements over a period are essential in assessing changes in growth rates. As a rough guide, the average weight of children can be taken as 3 kg (7 lb) at birth, 6.5 kg (14 lb) at 1 year, 13 kg (28 lb) at 2 years and 22.5 kg (49 lb) at 7 years

(the 'rule of seven' when using the old Imperial measure). However, it can be seen from the percentile charts that there is a wide range above and below the average.

In infants under the age of 2 years the head circumference should be measured. The standard measurement is the occipitofrontal circumference, which is the largest circumference of the head. The average head circumference at birth is 35 cm, at 3 months 41 cm, at 6 months 43 cm, at 1 year 46 cm and at 2 years 49 cm (Fig. 75). Hydrocephalus should be suspected when the rate of growth of the head is greater than is normal for the sex, age and size of the infant.

Figs 69–74 show standard height and weights for boys and girls aged up to 3 years, 2 to 10 years and 9 to 18 years. They have been modified from charts developed at the Institute of Child Health and are used by kind permission of J. M. Tanner and R. H. Whitehouse, Institute of Child Health and Hospital for Sick Children, Great Ormond Street, London. They include a page on which a child's growth and development can be recorded and full directions for the correct methods of measurement are also provided on each card.

Figs 75 and 76 show head circumference and skeletal maturity (or bone age), assessed by the state of fusion of the epiphyses.

The range of normal is expressed in percentiles and each chart shows the third, tenth, twenty-fifth, fiftieth, seventy-fifth, ninetieth and ninety-seventh percentiles. The meaning of the tenth percentile for height is that 10% of all normal children are shorter than this height at the age concerned.

The limits of normality to be accepted must depend on the purpose for which the charts are being used and on local conditions. As a rough guide, however, it can be said that children who fall outside the area between the tenth and ninetieth percentiles should be regarded with suspicion, and those outside the area between the third and ninety-seventh percentiles should be regarded as unhealthy unless proved otherwise.

Blood pressure

Abnormalities of blood pressure are uncommon in childhood, and measurement of blood pressure is a distressing examination for some young children. Consequently this procedure is sometimes omitted from the general examination, but it must be carried out in all cases of suspected cardiovascular disease. Blood pressure readings are best obtained in most children after the main examination is over and the child is partially dressed. Allow the child to see and play with the cuff and give some simple explanation of what is going to happen. The size of cuff is most important if accurate readings are to be obtained, and a variety of sizes are available. The inflatable bag should be long enough to encircle the full circumference of the arm and should be of a width roughly equal to half the length of the upper arm. In small children and infants it may not be possible to determine the blood pressure by auscultation, and the pulse can be palpated to obtain the systolic pressure. In babies

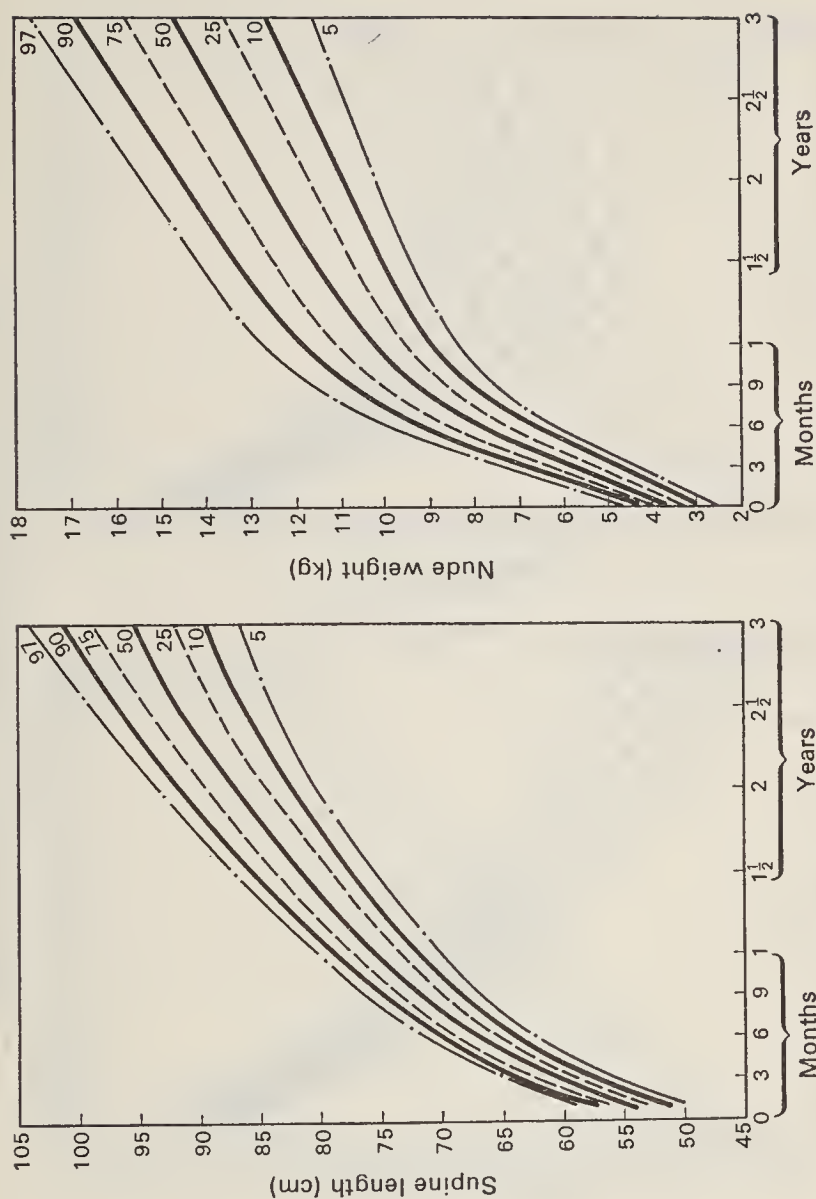


Fig. 69. Supine length and nude weight in boys up to 3 years.

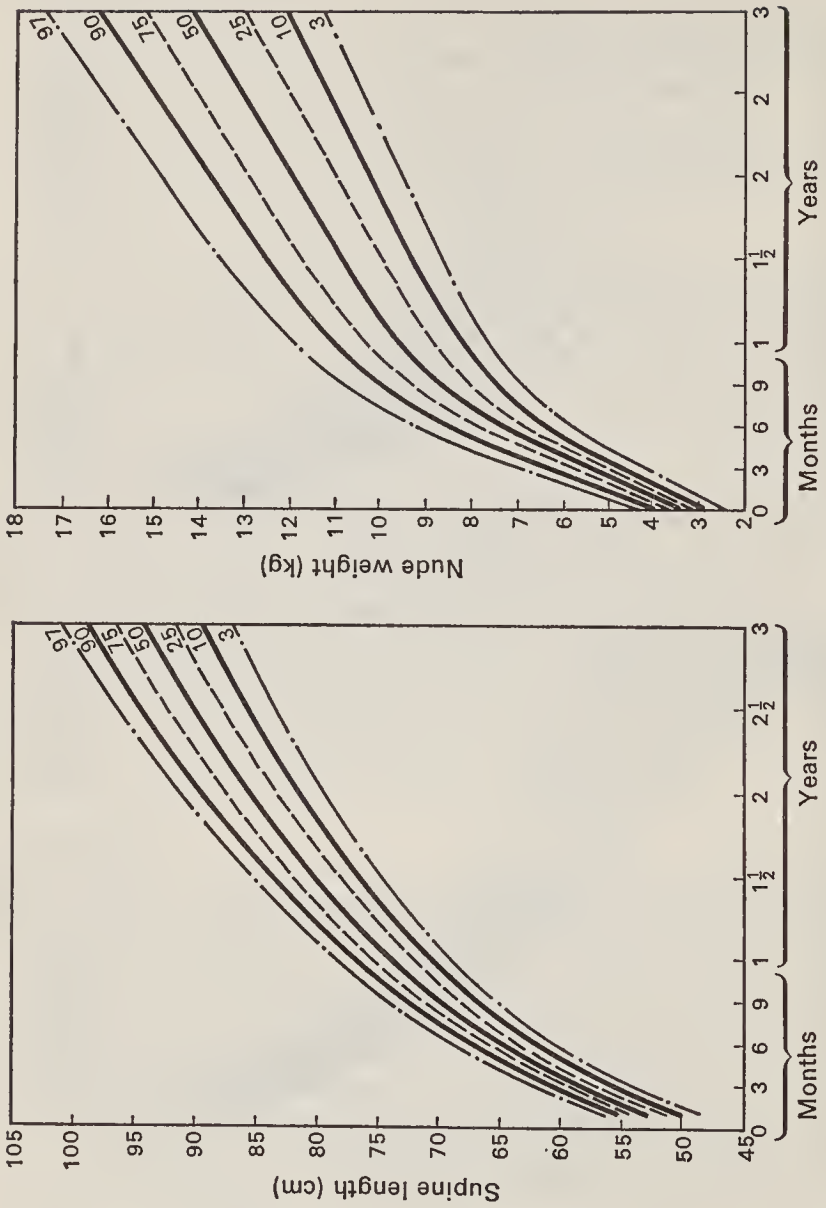


Fig. 70. Supine length and nude weight for girls up to 3 years.

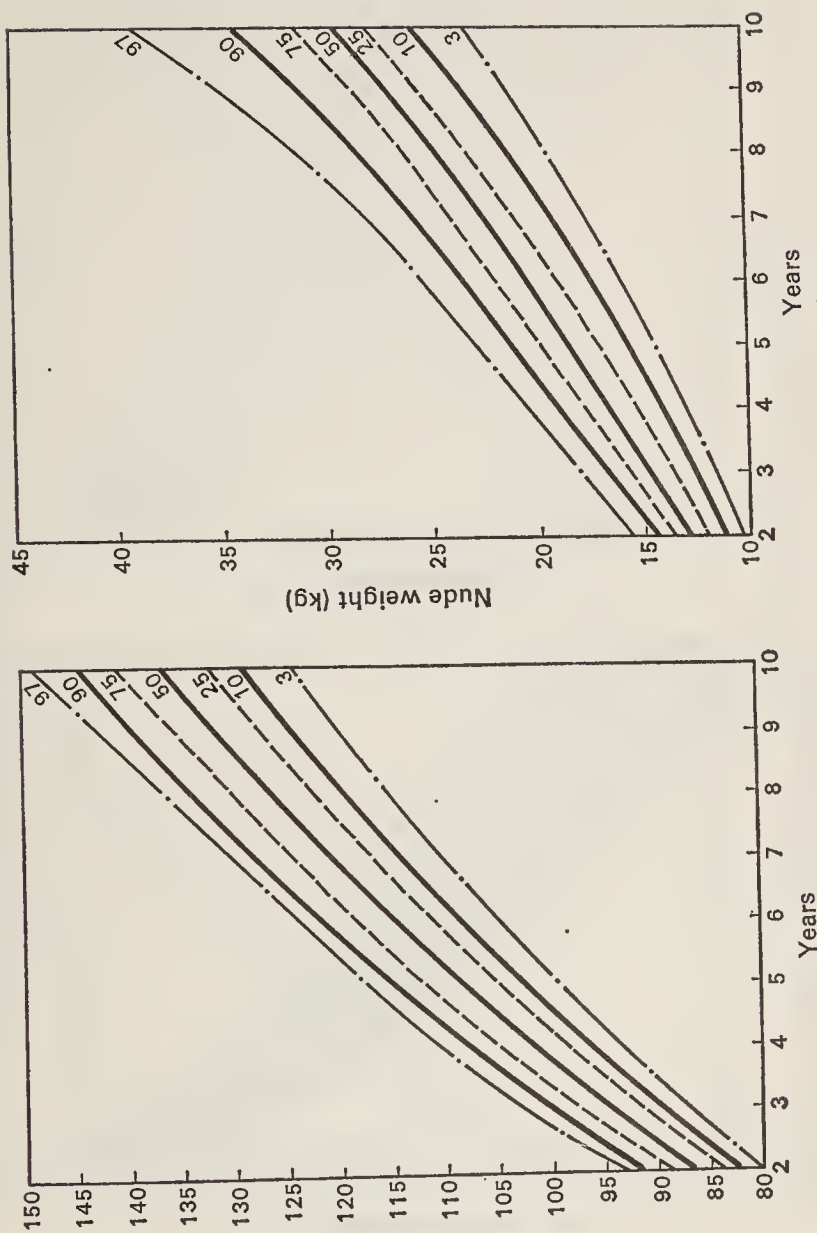


Fig. 71. Standing height and nude weight in boys aged 2 to 10 years.

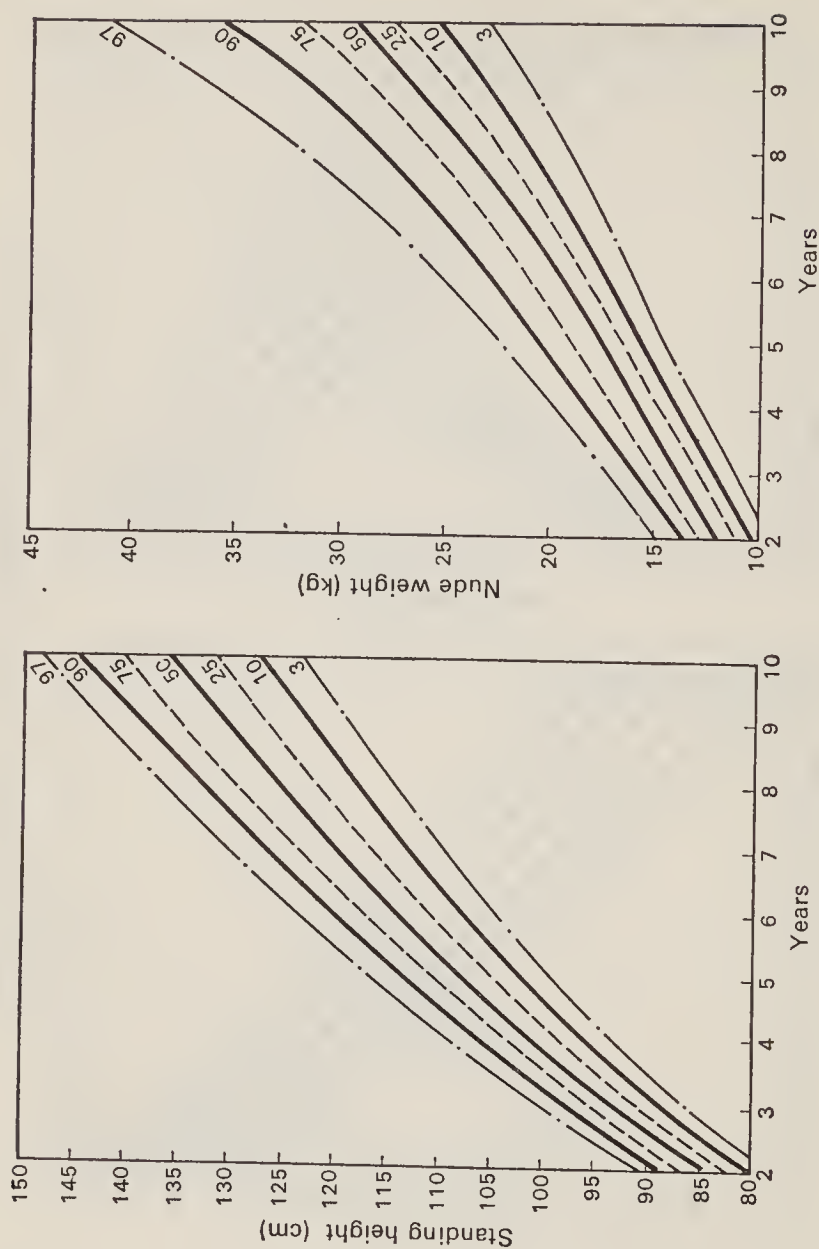


Fig. 72. Standing height and nude weight for girls aged 2 to 10 years.

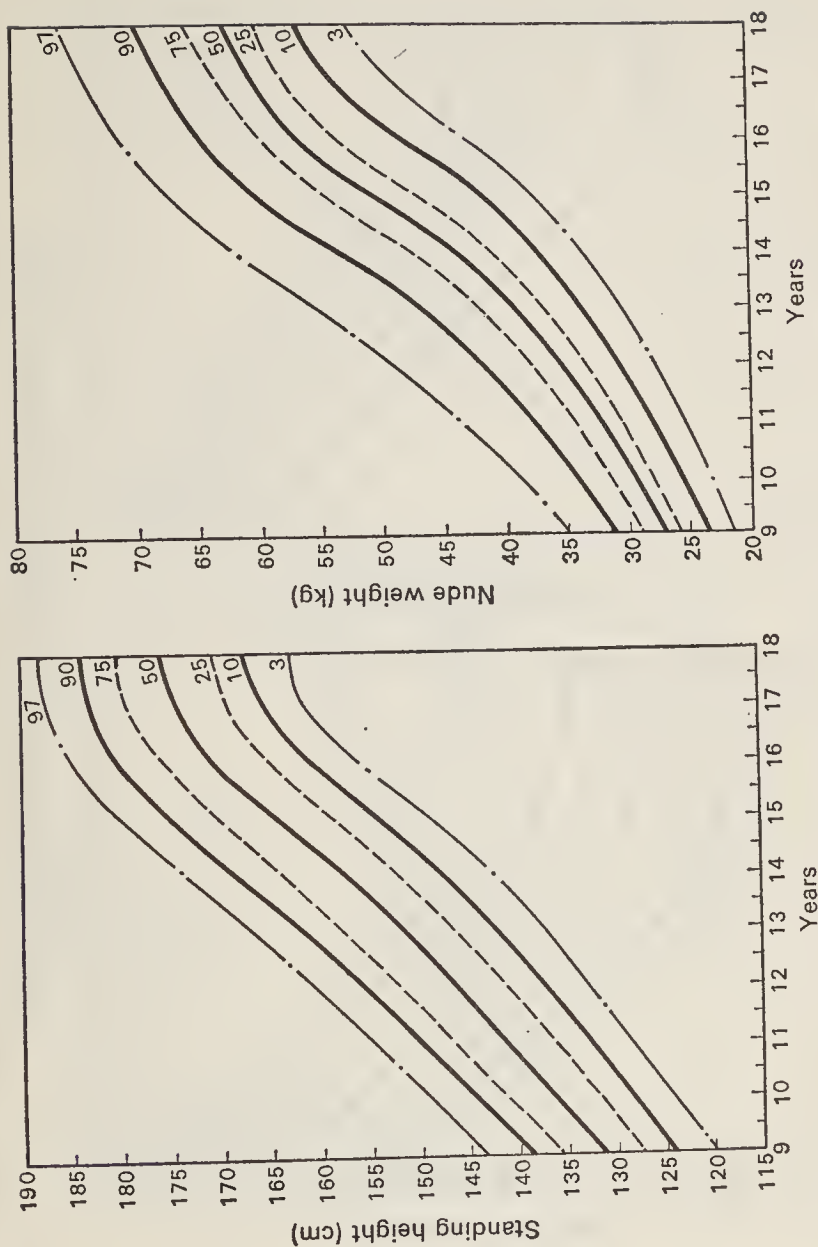


Fig. 73. Standing height and nude weight for boys aged 9 to 18 years.

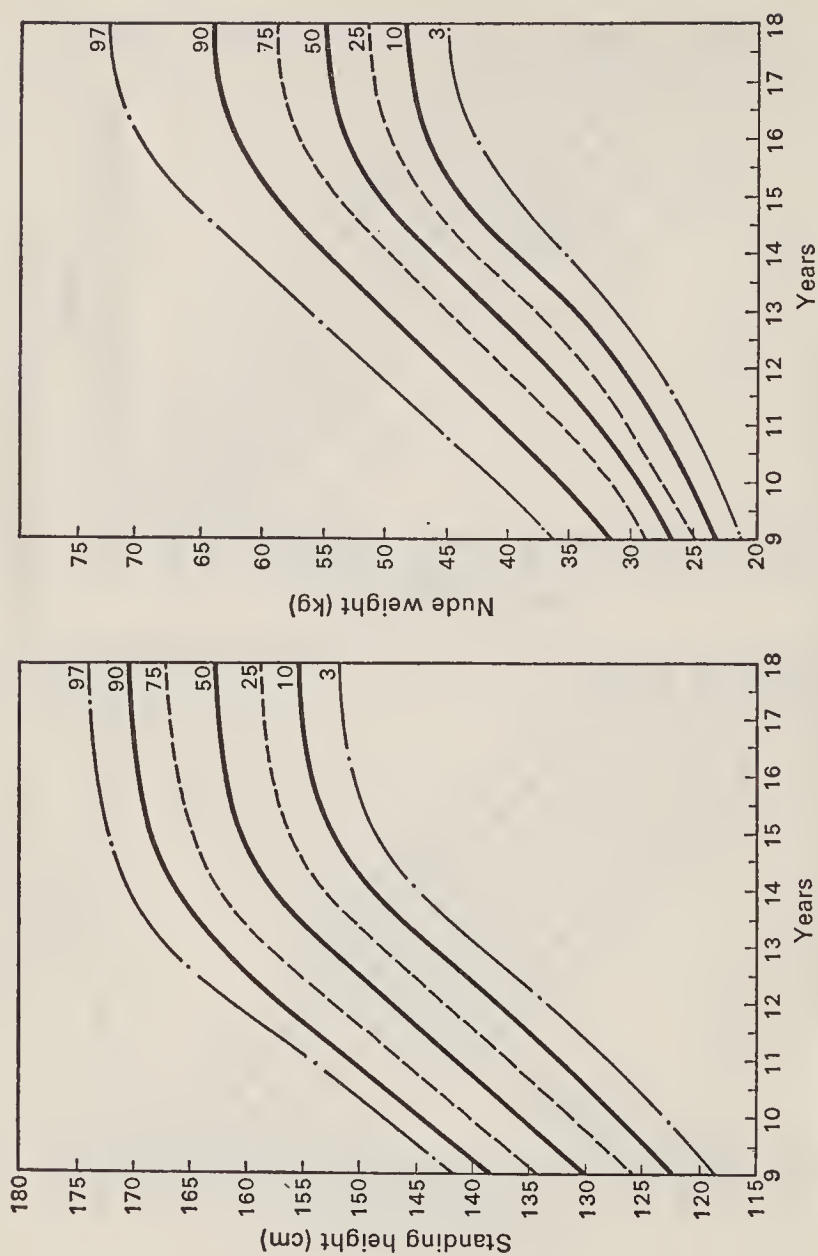


Fig. 74. Standing height and nude weight for girls aged 9 to 18 years.

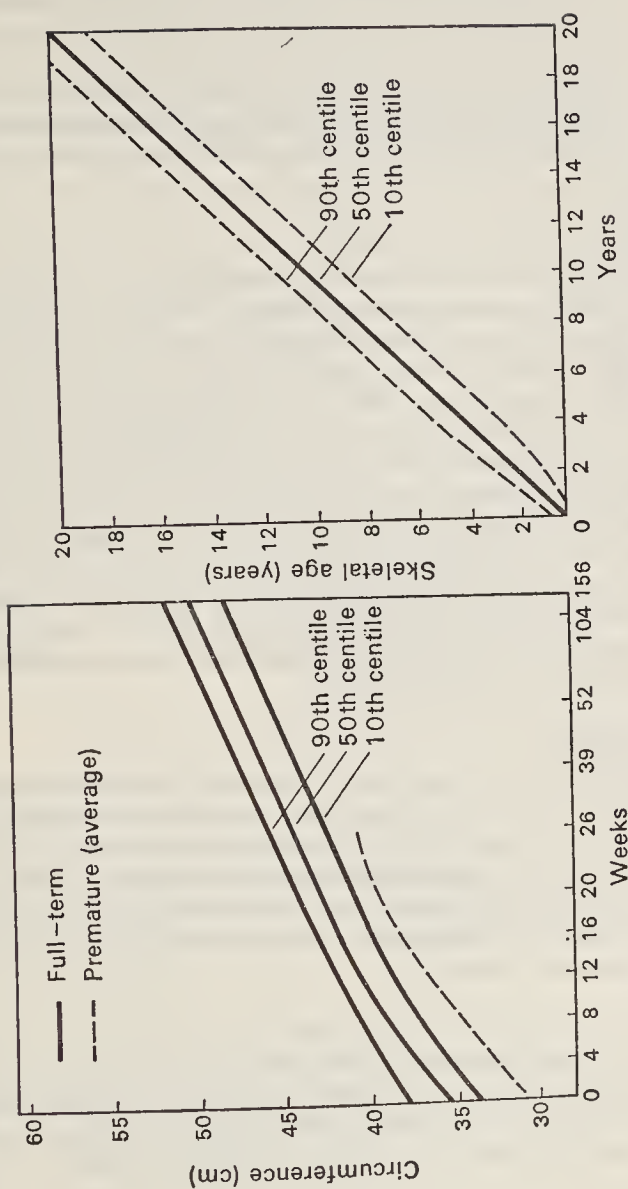


Fig. 75. Head circumference.

Fig. 76. Skeletal maturity.

the flush method may be used. The arm is held up and tightly bandaged to exclude the blood to the level of the cuff which is then inflated. The bandage is then removed to reveal a white limb. The pressure in the cuff is slowly reduced. The point at which the skin flushes is an approximate indication of the systolic pressure. The blood pressure in the legs must be determined in suspected cases of coarctation of the aorta. The average level of blood pressure in the arms is 80/50 mmHg in the newborn, 85/60 at 4 years, 95/65 at 8 years, 100/70 at 10 years and 110/75 at 13 years.

Temperature

The taking of the temperature is not an essential part of the examination of children. Fever is a very common finding in children and may be due to excitement, exercise and minor infections as well as to severe infections and other serious illnesses. Small infants often respond to infections with low temperatures. The temperature may be taken either before or after the physical examination. Oral temperatures are taken in children over the age of about 6 years. In smaller children, between 2 and 6 years, the thermometer can be placed in the axilla or groin. Rectal temperatures are taken in infants. The temperature in the axilla or groin is about 1°F (0.5°C) lower and in the rectum about 1°F higher than the oral temperature. The temperature of normal children varies between 1°F below and 1°F above the average. Rapid rises of temperature to 103° or 104°F (39.5° or 40°C) are sometimes associated with febrile convulsions in young children.

Rectal examination

Most children find rectal examination extremely unpleasant and small infants experience some pain from the procedure even when the little finger is used. One must use one's judgement, therefore, in deciding whether this examination should be inflicted on a child. Anal fissure is a common condition in childhood, and can be detected by close inspection of the anal canal, the buttocks and anal orifice being gently held open with one hand on either side.

Stools

The stools of normal breast-fed infants may be loose and green or pasty and yellow. Infants fed on cow's milk preparations pass stools of a paler yellow colour and of a much firmer consistency. The character of the stool in older children is more variable than in adults.

Urine

Special techniques are required for the collection of urine specimens in infants who have not yet acquired sphincter control. It may be possible

to 'catch' a specimen by holding the infant over a sterile container. Alternatively specially made sterile plastic containers with an adhesive opening can be applied to the washed genitalia. In the last resort bladder urine may be obtained by careful subrapubic aspiration.

Developmental screening examination

Development is the normal process of maturation of function which takes place in the early years of life. Disease and environmental disturbances commonly cause developmental delay and this can be detected by developmental examination. A simple developmental screening examination should be carried out at regular intervals on all normal infants and as part of the clinical examination on infants presenting with symptoms.

The main fields of development are:

1. Gross motor.
2. Vision and manipulation.
3. Hearing and speech.
4. Skills and social behaviour.

By using a table of average achievements in normal infants under each of these headings it is possible to detect delayed development. A representative selection of the stages of development are referred to as 'milestones'. The following is a list of milestones with the average age at which they are reached by normal children. There is of course a wide variation in the rate of development in individual children.

MILESTONES OF DEVELOPMENT

Smiles (6 weeks)

Holds head steady (3 months)

Turns head to sound (4 months)

Reaches out and grasps object (5 months)

Sits unsupported (7 months)

Transfers objects from one hand to the other (7 months)

Stands with support (9 months)

Crawls (11 months)

Says 2-3 single words with meaning (12 months)

Walks unsupported (13 months)

Holds cup and drinks (15 months)

Builds a tower of 3 cubes (18 months)

Says 2-3 word sentences (2 years)

Dresses himself (3 years)

EXAMINATION OF THE NEWBORN

Detailed examination of the newborn infant requires special techniques and skills. It is possible, however, to obtain important information by a few simple observations, which will now be described.

The newborn infant must be examined with all his clothing removed. Careful inspection will reveal any anatomical defects, such as cleft lip, abnormalities of the limbs or spina bifida cystica. In looking for anatomical defects, remember to examine the palate (for clefts), the anus (which may be imperforate) and the external genitalia. The testes can usually be felt in the scrotum in baby boys.

Observe the colour of the *skin*. Peripheral cyanosis is a common finding in newborns, but central cyanosis indicates cardiac or respiratory disease. So-called physiological jaundice is present after the first 48 hours in most premature babies and in some full-term babies. Severe jaundice within the first 48 hours is usually due to some haemolytic disorder. Look for haemangiomas in the skin. These may be flat capillary lesions, commonly seen on the eyelids and the back of the neck, or raised capillary-cavernous lesions (strawberry birthmark). In postmature babies the skin may be dry, cracked and peeling. Patchy papular lesions which fade rapidly are common and of no serious significance (erythema toxicum). 'Mongolian blue spot' is the name given to bluish areas of pigmentation in the sacral area, seen mostly in babies of African or Asian origin.

Breast enlargement, often with exudation of a milky fluid from the nipples, is frequently seen in newborn infants of both sexes. This is due to maternal hormones and is of no consequence.

The *head* should be inspected, palpated and measured (occipito-frontal circumference). There is considerable normal variation in the size of the fontanelles and in the width of the suture lines. A cephal-haematoma is a subperiosteal haematoma which appears a few days after birth as a large cystic swelling limited to the area of one of the bones of the skull vault. The caput succedaneum is an area of oedema of the scalp over the presenting part of the head. It pits on pressure and is not fluctuant.

It is essential to examine the *hips* of all newborn infants, in order to detect actual or potential dislocation. The infant is laid on his back with hips and knees flexed. The examiner grasps the thighs with the middle fingers over the greater trochanters and the thumbs over the lesser trochanters. Pressure with the fingers will replace a dislocated femoral head and pressure with the thumbs will dislocate an unstable joint. Both abnormal movements will be associated with a pronounced and palpable click.

Soft *systolic murmurs* are heard in many newborn infants. They are

usually of no significance and disappear after a few weeks but may, if they persist, indicate a congenital abnormality of the heart.

In the *neurological examination* note the power, tone and tendon reflexes in the limbs and look for obvious cranial nerve lesions such as facial palsy. Equally important are posture and the primitive reflexes. In either the prone or the supine position the limbs are normally held in flexion. There are a number of primitive reflexes, for example the finger grasp, the primary walking and the Moro (startle) reflexes which are present in normal full-term infants and disappear after about 3 months. The absence of these reflexes, especially if associated with abnormal tone and posture, suggests a lesion of the central nervous system. The persistence of primitive reflexes beyond the normal time of disappearance is also an abnormal finding suggestive of cerebral palsy.

13

COOPERATION WITH LABORATORIES

*What Investigations to Request — What Information to Send — Making the
Best Use of Results — Collection of Specimens*

Cooperation with laboratories requires a degree of mutual understanding between clinicians and laboratory staff of all grades, that is sometimes less evident than it should be. Pathology is nowadays a highly specialized subject, usually divided into at least four sub-specialities. Clinicians cannot be expected to be familiar with details of the performance of the investigations that they request, but they should know:

1. What *investigations* to request in given circumstances.
2. What *information* the pathologist will require about the patient.
3. How to make the *best use* of the results received from the laboratory.
4. What *specimens* the pathologist will require and how they should be transmitted to the laboratory.

In the case of more complicated investigations much waste of time and effort (let alone discomfort to the patient) would be avoided if the clinician and pathologist concerned could, whenever possible, discuss the problem to be solved before a programme of investigation is embarked on: and in the case of more complicated investigations clinicians should certainly consult the pathologist concerned about the interpretation of results. Young doctors are occasionally heard to say that they have *ordered* or neglected to *order* a particular investigation. This unfortunate term suggests an attitude to the laboratory which is hardly likely to be conducive to cooperation. One does not *order* a colleague to perform what may be a highly skilled piece of investigation. One *requests* him to do this, and one must then cooperate in every way possible.

WHAT INVESTIGATIONS TO REQUEST

This is a more complicated question than it may at first appear and depends on a number of factors, including the laboratory resources available (and particularly how far these are automated) and, in the case of many hospitals today, their policy as regards bed occupancy.

A laboratory result is one part of the investigation of a patient and has just as much or as little significance as any other physical finding. Such a result may suggest an unequivocal diagnosis, just as physical finding may do so, or it may have very little significance.

In the past it was customary to perform a reasonably complete physical examination on every patient, including the examination of systems not obviously implicated: but it was customary to use special investigations selectively, requesting only those which appeared likely to throw relevant light on the problem in hand. This policy was in the main dictated by the laboratory resources available and it must continue at least to some degree where laboratories are manually operated, if such laboratories are not to become overloaded and consequently less efficient.

But with increasing automation, particularly in haematology and biochemistry, the position is changing rapidly towards a policy of performing all automated routine investigations on all patients; for where a machine is set up to perform a number of routine investigations on one sample of blood, for example, it is neither sensible nor economic to request only one or two of these investigations.

Further if the hospital needs above all to have a quick bed turnover, it may be uneconomic not to request as many as possible of the investigations that are likely to be necessary as soon as the patient is admitted.

It is therefore now impossible to lay down rules about what to request in given circumstances. Junior doctors should make themselves aware of their seniors' policy on such matters and this is one of the situations where overall policy must be decided by consultation between clinicians and pathologists.

The authors of this book do not retract from their own opinion expressed in the first chapter, that *no doctor should request an investigation unless he knows what information relevant to the problem the investigation is likely to provide*; for they believe that the discipline of this approach is essential for the doctor's continuing education, however much investigations may be automated. The same approach is of course even more important in case of more complicated procedures which are not and are unlikely to become automated. Doctors should also be aware of *possible risks to the patient* and should have some idea of the *cost* of the investigations that they request.

WHAT INFORMATION TO SEND

Forms of requests for pathological investigations must be filled up completely, accurately and legibly. It is important to include the patient's surname and first name, his address (or ward and bed number if in hospital), his hospital serial number and his date of birth. These

details are particularly important in areas where many names are very similar, as for instance in Wales and some eastern countries. Requests must also indicate the exact nature of the material sent, its source, its date and time of removal and, of course, the nature of the investigations requested. In the case of patients who are not in hospital the name, address and telephone number of the doctor making the request must be included.

It is important to realize, however, that telephoning laboratory results is a procedure fraught with danger and to be avoided if possible. If it has to be done the recipient of the message must write it down (and not trust to his memory) and repeat it back to the laboratory worker who should check its accuracy. A confirmatory written report should always follow a telephone message.

It is generally desirable, and essential in the case of many investigations, that the request should give a note of the duration of the illness, the tentative diagnosis and the clinical state of the patient. A note of any current antibiotic therapy is essential with all requests for bacteriological examinations. It is known, too, that some drugs seriously interfere with some laboratory estimations. Examples are the effect of X-ray contrast media on the estimation of PBI and the effect of barbiturates on urinary steroids. It is likely that other examples will be found and it is thus desirable that all requests should include a note of current therapy.

MAKING THE BEST USE OF RESULTS

The 'normal range'

Laboratory results, especially in haematology and biochemistry, are usually expressed quantitatively in relation to a so-called 'normal range'. If any single measurement is made on, say, 1000 symptomless apparently healthy people and the results are plotted as a graph, a curve like those illustrated on p. 136 for the diameter of red blood cells will be obtained. A few people will have values above or below those of the great majority. By convention the 'normal range' is usually quoted as the range into which over 80% of the results on symptomless apparently healthy people fall. The normal value of the blood urea for example is usually given as 20–40 mg/100 ml. A level of 50 mg probably indicates mild urea retention, but this result might be found in one of the few people whose normal level falls outside the 80% range.

It is important to realize, too, that the 'normal range' may depend on the method used and so may vary from laboratory to laboratory and may also vary to some extent with age, sex, pregnancy, occupation, race and medication, including oral contraceptives. Small deviations from the 'normal range' do not necessarily indicate disease.

Laboratory errors

Quantitative measurements are always subject to error. It may be said, however, that given a good laboratory service *serious errors are usually due to the bad labelling of specimens or mistakes in the transmission of results*, rather than to errors arising actually in the laboratory.

The introduction of modern automated methods, in which results are calculated and printed out by the computer, may give a false sense of security. Small constant errors can occur, as can occasional 'rogue results'. If a quantitative result from any source, manual or automated, appears contrary to what might be expected the investigation should be repeated on a fresh specimen, if necessary on several occasions.

It need hardly be said that the wise pathologist keeps a constant eye on the results going out from his laboratory and the wise clinician immediately consults the pathologist if any difficulty arises about the interpretation of results.

COLLECTION OF SPECIMENS

It is essential that specimens reach the laboratory fresh and correctly taken into the correct kind of containers. Specimens are best taken by hand to the laboratory as soon as they are obtained, but they can, if if necessary, be sent by post (letter post only), provided they are suitably packed and labelled 'Fragile, With Care' and 'Pathological Specimen'. Such specimens must be placed in a sealed inner container and then packed in a wooden or metal box containing sufficient absorbent material to soak up all the liquid contents if the inner container is broken. Local and international regulations about the transmission of pathological material must be strictly adhered to.

Suitable containers are best obtained from the laboratory that is going to make the investigations. All containers must be perfectly clean and preferably sterile. This is of course essential for bacteriological specimens. Containers for blood should be completely dry. All containers must have properly fitting lids or caps. It is usually essential that the correct container should be used for each particular investigation (e.g. particular anticoagulants are necessary for particular chemical or other tests on blood). It is also desirable that the amount of material specified for a particular container should be placed in that container, and in any case no more than the amount specified.

All syringes and needles must be sterile and should either be of the disposable variety or be dry-sterilized in a laboratory.

Venepuncture

A piece of rubber tubing is used as a tourniquet and applied round the upper arm over the middle of the biceps, so as to impede the venous but

not the arterial flow. The skin at the bend of the elbow is 'painted' with 0.5% chlorhexidine in 70% alcohol or simply 70% spirit (iodine is expensive and can give rise to severe skin reactions). The skin is rendered tense by the operator's left hand; the syringe with the needle attached is held in the right hand and almost parallel with the patient's arm; the patient is asked to 'make a fist' and then the needle with the bevel upwards is inserted into a prominent vein—the median basilic is usually convenient—and the needle is pointed in the direction of the blood flow. The required amount of blood is then drawn up into the syringe and the tourniquet is removed before the needle is withdrawn, as otherwise a haematoma may form. For some purposes it is necessary to remove the tourniquet as soon as the needle enters the vein, so that free-flowing blood is withdrawn. As soon as the needle is withdrawn a swab is placed on the puncture site and the patient is instructed to hold his forearm firmly flexed against his arm for a minute or so. Occasionally a vein in the forearm or wrist may prove more convenient than one at the elbow, but the procedure is then usually more painful. A vein which can be *felt* is generally easier to enter than one which can only be *seen*.

Blood obtained by venepuncture should be placed immediately in a container suitable for the purpose for which it is to be used. The needle should first be removed from the syringe, since forcing the blood through the needle may cause haemolysis. Appropriate containers for particular investigations should be obtained from the laboratory, since, when unclotted blood is required different anticoagulants are needed for particular purposes.

Heparin and sequestrene (EDTA) are the most generally useful anticoagulants. Sequestrene can be used for most haematological investigations and heparin for most simple chemical tests, with the exception of blood glucose for which special bottles containing sodium fluoride are necessary.

For blood groups and serological investigations blood should be taken into a dry sterile bottle or tube. If the specimen has to be sent to the laboratory by post, it is best to wait till the blood has clotted. Some serum should then be removed with a sterile needle and syringe, and this serum should be sent separately, together with the blood clot.

Lumbar puncture

See p. 259.

Sterile urine

See p. 86.

There follow Tables 10 to 13, dealing with microbiology, haemato-

logy, immunology and chemical pathology; these give information about what specimens to send and how to send them, with some brief notes about some of the more important investigations, which may be requested. The tables are necessarily incomplete and different laboratories may have different requirements as regards specimens. *They therefore in no way reduce the need for consultation with the pathologist to whom specimens are sent or for reference to specialized text books of pathology.*

TABLE 10. MICROBIOLOGY

Tests	Specimen required	Notes
Blood culture	10 ml of whole blood, taken with strictest aseptic precautions into sterile glass container (special blood culture bottle if possible), preferably when patient's temperature is rising and before administration of antibiotics. For this investigation only, blood should be squirted through needle into container. Several specimens should be sent	<p>Aerobic and anaerobic culture should be requested. Sensitivity of organisms to antibiotics may be determined and quantitative cultures may be performed. Useful mainly in the investigation of pyrexia of undetermined origin (PUO) and in diagnosis of causative agent in subacute bacterial endocarditis. The <i>most important</i> conditions in which bacteraemia and a positive blood culture may be found are:</p> <p>Pyogenic infections due to streptococci, staphylococci, pneumococci and meningococci</p> <p>Typhoid fever { in first 7-10 days</p> <p>Paratyphoid fever { days</p> <p>Brucellosis</p> <p>Leptospirosis</p> <p>Gram-negative bacteraemia (<i>Escherichia coli</i> and <i>Pseudomonas pyocyanea</i> are occasionally found in blood stream)</p>
Serum tests	5 ml whole blood placed in sterile container and allowed to clot. If specimen is to be posted, allow blood to clot, remove some serum with a sterile syringe and needle and send serum and clot in separate containers	<p>The most important <i>agglutination</i> tests are:</p> <p><i>Widal reaction</i> for typhoid and paratyphoid fevers. Become positive in second and third weeks. Rising titre important. Low titres may be found in normal persons and higher ones in patients recovered from enteric infections and in persons who have received TAB. In the latter the H antigen usually predominates. Vi antigen is found in carriers.</p> <p><i>Brucellosis</i>. Test usually positive after first week, but antibody production very variable.</p>

Paul Bunnell test for glandular fever. Diagnostic in titres over 1/512. Consult pathologist about lower titres

The most important *complement fixation tests* are those used in the diagnosis of syphilis (see below) and of virus infections (see p. 335). A variety of CFT tests is available for diagnosis of parasitic infestations, including amoebiasis and hydatid disease

Tests for syphilis 10 ml whole blood allowed to clot

The available serological tests for treponemal infection fall into two groups:

1. *Reagin detectors* (Standard Tests, STS). These detect a non-specific antibody-like reagin, which is produced in syphilis; but also in autoimmune diseases, collagen diseases, leprosy and after inoculations, so that biological false positive reactions occur

- a. *Complement Fixation Tests*: WR: Cardioliolin W.R. (CWR)

- b *Flocculation Tests*: Kahn: Price's Precipitation Reaction (PPR): Venereal Disease Research Laboratory slide test (VDRL): Rapid Plasma Reagin card test (RPR) and Automated Reagin Test (ART)

2. *Treponemal tests*: These detect antitreponemal antibodies and, though more specific than reagin tests, may all be positive in other treponemal infections e.g. yaws.

Treponemal Immobilization (TPI)

Reiter Protein Complement Fixation Test (RPCFT)

Fluorescent Treponemal Antibody (FTA 200): serum is diluted 1:200 to obviate non-specific group reactions.

Fluorescent Treponemal Antibody Absorbed (FTA-ABS): group antibody is removed by absorption so that serum can

TABLE 10. MICROBIOLOGY—(cont.)

Tests	Specimen required	Notes
Pus and sero- purulent discharges	Specimen should be sent in a wide-mouthed sterile container or on a throat swab contained in a glass test tube. Care must be taken not to contaminate the outside of the container. Specimens should be collected if possible before treatment. If not so collected a note of any antibiotic treatment must be sent. If gonorrhoea is suspected swabs should be sent in Stuart's transport medium	<p>be tested in 1:5 dilution, <i>Treponema Pallidum Haemagglutination</i> (TPHA)</p> <p>FTA-ABS and TPHA are the most sensitive serological tests for syphilis, but are only available in special centres.</p> <p>For ordinary purposes a positive reagin test together with a positive RPCFT makes a diagnosis of syphilis highly likely. These latter tests become positive 1-3 weeks after infection and may be altered by treatment. They are positive in all untreated secondary syphilis and in most cases of untreated tertiary. The more sensitive tests (FTA-ABS and TPHA) become positive earlier and tend to remain positive despite effective treatment</p> <p><i>Treponema pallidum</i> can usually be detected by dark ground microscopy or fluorescent techniques in fluid from early lesions or from lymph nodes by gland puncture</p> <p>The most important organisms found are:</p> <p>Staphylococci Streptococci Pneumococci <i>Mycobacterium tuberculosis</i> Gonococci Meningococci <i>Escherichia coli</i> <i>Actinomyces</i> (sulphur granules may be seen in pus) Organisms causing gas gangrene (e.g. <i>Clostridium welchii</i>)</p>

Urine

For collection of specimens see p. 86. The use of catheters should be avoided. Send at least 50 ml in sterile container with minimum delay. If delay inevitable, cool specimen to 4°C in refrigerator

Faeces

Send either

1. Whole stool passed into a clean bed pan
2. An aliquot portion in a sterile container, carefully sealed to prevent outside contamination
3. A swab dipped into a freshly passed liquid stool. This is preferable to a rectal swab. It is useless to send a rectal swab with no visible sign of faeces. In all cases specimen should be uncontaminated by urine or disinfectant

Cerebrospinal fluid (See also p. 260)

Send two specimens of not less than 5 ml each in two sterile containers

For interpretation of bacteriological results see p. 87

Deposit is examined fresh for red cells, white cells, casts and crystals and in preparations stained by Ziehl-Neelsen's method for tubercle bacilli. If pus present in apparently sterile urine and no tubercle bacilli are found by this method, repeated early morning urines should be cultured for tubercle bacilli

The parasites found in the faeces are described on pp. 65-71

Stools for *Entamoeba histolytica* should reach the laboratory while still warm (p. 68)

The important pathogenic bacteria of the stools are those of the enteric and dysentery groups, which are identified, after their isolation in pure culture, by fermentation and agglutination tests. Carriers may be detected in this way, but repeated examinations may be necessary

In suspected meningitis microscopical, bacteriological and chemical examination is necessary. Stained films may show tubercle bacilli. Cultural and inoculation methods for tubercle bacilli take too long to be of practical value in the initial diagnosis. A lymphocytic fluid which contains half the glucose concentration of the blood in a case of meningitis should be taken as indication for starting and completing course of treatment for tuberculous meningitis

TABLE 10. MICROBIOLOGY—(cont.)

Tests	Specimen required	Notes
Throat and other swabs	Swabs are best purchased in bulk readily sterilized. 'Home made' cotton-wool swabs must be dipped in bovine albumen and steam sterilized before use. No antiseptic gargle or other preparation should have been used for at least 3 hours. Rub the swab firmly over any exudate which may be seen and return it immediately to its protective tube (or break	<p>The other <i>important</i> bacteria found in patients presenting with meningitis are meningococci, pneumococci, streptococci, staphylococci, and <i>Haemophilus influenzae</i></p> <p>In meningitis secondary to head injuries and neurosurgical operations a variety of organisms, including Gram-negative ones may be found. For virus meningitis see p. 335</p> <p>Usually fatal meningo-encephalitis has been described due to infection via the respiratory tract with free living soil amoebae of the genera <i>Hartmannella</i> (or <i>Acanthamoeba</i>) and <i>Naegleria</i> (an amoeboflagellate). Their trophozoites can be seen in a small drop of CSF sediment examined fresh under the microscope with diminished light or by phase microscopy; and can be cultured by special methods. The disease is acquired through the contamination of the respiratory tract, usually by swimming in infected water in warm climates</p> <p>Cultures from throat swabs are important to confirm a <i>clinical diagnosis of diphtheria</i>, but if clinical picture is present, antitoxin should be administered without waiting for bacteriological confirmation. Other organisms, including those of Vincent's angina, may be identified in stained films or by cultural methods.</p> <p>Healthy carriers, e.g. of streptococci in the throat or of staphylococci in the nose, may also be identified in this way</p> <p>For vaginal swabs see p. 50</p>

Swabs for gonococci and *Trichomonas* should be send in Stuart's transport medium

wooden stem and drop end into sterile container). The swab should be taken under direct vision with good lighting. If the patient is a child, the doctor or nurse should wear a face-mask. Small children should be held firmly by a nurse or parent

Sputum

If the sputum is copious, send a few ml in a wide-mouthed sterile container. If it is not copious, explain to the patient that what is wanted is something from 'deep in the chest'. This should prevent the collection of specimens of saliva, rather than sputum. Except when looking for tubercle bacilli, it is usually fruitless to send sputum for culture unless it looks purulent to the naked eye

Tubercle bacilli may be seen in appropriately stained films, but repeated specimens should be examined. Culture for tubercle bacilli takes up to 6 weeks. Other organisms may be identified by staining or cultural methods and sensitivity to antibiotics determined. Malignant cells and asbestos bodies (Plate XII) may be demonstrated by special staining techniques. *Aspergillus fumigatus* can be cultured on appropriate media

Virus infections

Most virus techniques are only available in specialized laboratories. Therefore doctors requiring their aid should get in touch with the specialist in charge of such a laboratory. Four special problems are mentioned here

Meningitis

Send CSF blood, faeces and a throat swab, taken with strict sterile precautions and then packed with ice in a vacuum flask. If more than 12 hours likely to elapse, specimens must be packed in solid carbon dioxide with special precautions. Use wooden swab and break off tip into sterile container

Virus investigations indicated when patient with meningitis has lymphocytic CSF without reduction in glucose concentration. Send further blood sample in 5 days' time

TABLE 10. MICROBIOLOGY---(cont.)

<i>Tests</i>	<i>Specimen required</i>	<i>Notes</i>
Respiratory disease	Send sputum, throat swabs or washings and serum, packed as above	Send further specimen of serum 7 days later
Suspected smallpox	<p><i>Papular</i>: Scrape lesions with a needle and make smears on clean glass slides. Allow smears to dry without heat</p> <p><i>Vesicular</i>: Collect fluid in capillary tubes from 6-10 lesions</p> <p><i>Crusting</i>: Twelve scabs should be removed and placed in small sterile bottle. 5 ml of whole blood should also be sent</p>	Immediate report may be available if characteristic particles are found on electron microscopy. Antibody diffusion may give result in 2-6 hours. CF techniques may give result in 24 hours, if adequate material available to prepare antigen. Egg inoculation may give result in 3-6 days
Rubella	5 ml of whole blood as above and further specimen in 2 weeks' time	Especially important in first trimester of pregnancy. A rise in antibody titre is good evidence of rubella infection, and may detect subclinical attacks. Susceptibility to rubella virus can also be demonstrated
Other viral studies	10 ml whole blood as above with further specimen in 10-14 days' time	Many sophisticated techniques are in use. Of particular importance to clinicians are serum tests for Q fever, psittacosis, lymphogranuloma inguinale, influenza A, B and C, adenoviruses, mumps, lymphocytic choriomeningitis and primary herpes simplex

TABLE 11. HAEMATOTOLOGY

<i>Investigation</i>	<i>Normal range</i>	<i>Specimen required</i>
Red cell count	Adult males 5500 000/ μ l (4500 000 to 6500 000) Adult females 4800 000/ μ l (3900 000 to 5500 000)	4 ml whole blood collected into sequestrene, heparin or ammonium potassium oxalate bottle. Sequestrene is the anticoagulant of choice (1 mg to 1 ml of blood)
White cell count	7000/ μ l (4000 to 11 000)	
Haemoglobin	Adult males 13.5 to 18 g/100 ml Adult females 11.5 to 16.5 g/100 ml	
Packed cell volume (PCV)	Males 47% (40 to 54) Females 42% (36 to 47)	
Mean corpuscular volume (MCV)	86 μ m ³ (76 to 96)	
Mean corpuscular haemoglobin (MCH)	29.5 pg (27 to 32)	
Mean corpuscular haemoglobin concentration (MCHC)	33% (30 to 35)	
Differential count	See p. 143	
Reticulocytes	0.2 to 2.0%	
		Fresh films (see p. 134). Satisfactory films can also be made for sequestrene blood up to 1 hour after collection. Films from blood left longer or from heparinized blood are less satisfactory

TABLE 11. HAEMATOTOLOGY—(cont.)

<i>Investigation</i>	<i>Normal range</i>	<i>Specimen required</i>
Platelets	150 000 to 400 000	
Osmotic fragility of red cells	Haemolysis begins at 0.5% saline and is complete at 0.32% saline (without incubation)	Can be performed on sequestrene or heparin specimen, as above, up to 8 hours after collection
Bleeding time	See p. 146	
Prothrombin time	10 to 14 seconds	5 ml whole blood in sodium citrate bottle. Note that prothrombin time is useless in patients on heparin therapy
Other coagulation studies	—	See p. 146
Coagulation time	Lee & White 11 minutes Dale & Laidlaw 3 minutes	
ESR	Westergren: Men 3–5 mm in 1 hour Women 4–7 mm in 1 hour Wintrobe: Men 0–9 mm in 1 hour Women 0.20 mm in 1 hour	2.5 ml whole blood in citrate bottle. Tests can be performed up to 3 or 4 hours after collection
Serum iron	80–180 $\mu\text{g}/100\text{ ml}$	
Total iron binding capacity	250–400 $\mu\text{g}/100\text{ ml}$	10 ml whole blood in heparin bottle
Serum folate	6–21 ng/ml	Require unhaemolysed blood in clean <i>new</i> glass container. Patient should have fasted for 6–8 hours and not be on antibiotics. Send details of all drug therapy. Consult laboratory concerned about requirements and normal range
Serum B12	150–1000 pg/ml	
Blood grouping and serology (including Coombs' test)	See pp. 149, 152	5 ml whole blood in dry tube, allowed to clot. If delay likely remove serum as on p. 328

TABLE 12. IMMUNOLOGY

Clinicians with no special training in immunology should *always* consult the pathologist in charge of the immunological laboratory about the problem to be solved, the investigation to be performed and the interpretation of the results of the investigations. Investigations most generally performed fall into three groups:

<i>Tests</i>	<i>Specimen required</i>	<i>Notes</i>
To assess B cell function and status	5 ml whole blood in sterile dry container should be taken immediately to the laboratory. If delay is inevitable serum should be separated as on p. 328, placed in the refrigerator while waiting and then be sent as quickly as possible to the laboratory, where it is finally stored at -20°C or below	Serum is suitable for: 1. Examination for all types of antibodies, including hetero- and autoantibodies. 2. Analysis by electrophoresis, immunoelectrophoresis and estimation of immunoglobulins
To assess T cell function and status	For practically all tests of cell mediated immunity <i>arrangements must be made in advance with the specialist laboratory concerned</i> . The patient will have to attend in person, so that a fresh specimen of blood can be processed in a manner appropriate for the particular test. The patient may also be required to attend a suitable clinic or laboratory for skin tests of CMI, e.g. tuberculin, candida, DNCB	Freshly isolated lymphocytes can be investigated for various functions, e.g. blast transformation, lymphokine production, e.g. MIF (macrophage inhibition factor) toxicity. In addition the proportions of T & B cells can be estimated
Examination of biopsies	Biopsy material, e.g. kidney or skin, requires immediate processing, usually cryofixation, within a few minutes of collection. It should therefore either be appropriately fixed at the bedside or in the theatre, or taken <i>immediately</i> to the laboratory for processing	Tissues can be examined for the presence of various immunoglobulins, complement and other proteins in specific sites

TABLE 13. CHEMICAL PATHOLOGY

Blood analysis. Laboratories use different methods and the specimens required for different determinations vary. Most single determinations can be made on the serum or heparinized plasma derived from 5 ml of whole blood, though for a few purposes whole blood itself is required (see below). Often a number of determinations can be made on one such sample and modern automated methods enable a large number of determinations to be made on much smaller samples. The laboratory concerned should be consulted and, where possible, special containers for different determinations should be collected from this laboratory.

Serum. 5 ml of whole blood should be placed in a dry sterile container and allowed to clot. If there is to be delay in delivery of the specimen to the laboratory, the serum should be removed with a sterile syringe and needle and placed in a separate container.

Plasma. Heparin should be used as the anticoagulant, except where otherwise stated. If delay is inevitable the anticoagulated blood should be centrifuged and the plasma be placed in another container, as for serum above. Freshly separated plasma is essential for sodium, potassium, chloride, bicarbonate and inorganic phosphate. For plasma cortisol determinations plasma must be separated immediately.

Whole blood for sugar or reducing substances must be anticoagulated with fluoride-oxalate mixture: for lead with heparin and for fibrinogen with plain oxalate.

Urinalysis. Specimens required vary with different laboratories and according to the purpose of the investigation (e.g. random sample for simple qualitative tests: measured aliquot when the concentration of a constituent is required or total 24 hour specimen for 24 hour excretion values). Different laboratories use different preservatives such as toluene, chloroform, thymol, methiolate or hydrochloric acid.

Radioimmunoassay is being used increasingly for the measurement of peptide hormones (e.g. growth hormone, ACTH, pituitary gonadotrophic hormones, TSH, gastrin insulin and glucagon) and some other substances. It is usually important that determinations are made under standard physiological conditions; and in some cases provocation tests are employed. The laboratory concerned should always be consulted about these determinations.

Drug assays are used for the diagnosis in cases of overdosage and most laboratories have facilities for blood salicylates and blood barbiturate. Send 10 ml heparinized whole blood.

The estimation of other drugs, usually for the monitoring of treatment e.g. phenytoin, quinidine and digoxin (the latter by radioimmunoassay), are only available in special centres.

Normal Values are expressed in the familiar mg/100 ml and also, according to the international system of units (SI units), in moles or subunits of moles, per litre (1 mole = 1 gram molecule). Students and doctors will have to become familiar with these units, which provide a uniform system for expressing physiological concentrations of both ions and unionized compounds, thus allowing the osmolality of a fluid of known composition to be calculated readily.

TABLE 13. CHEMICAL PATHOLOGY: BLOOD—(cont.)

	<i>Normal range</i>	<i>Specimen required</i>
Sodium	137–144 mmol/litre	Plasma freshly separated
Potassium	3.5–4.8 mmol/litre	Plasma freshly separated
Chloride	96–108 mmol/litre	Plasma freshly separated
Bicarbonate	23–28 mmol/litre	Plasma freshly separated
Calcium	8.8–10.6 mg/100 ml 4.4–5.3 mmol/litre	Plasma or serum*
Magnesium	1.8–2.4 mg/100 ml 0.7–1.0 mmol/litre	Plasma or serum
Inorganic phosphate	2.5–4.5 mg/100 ml 0.8–1.4 mmol/litre	Plasma freshly separated
Copper	80–150 µg/100 ml 13–24 µmol/litre	Serum
Lead	<40 µg/100 ml <1.9 µmol/litre	Serum or anticoagulated (heparin) whole blood
Creatinine	0.7–1.4 mg/100 ml 62–124 µmol/litre	Plasma or serum
Uric acid	2.7 mg/100 ml 0.12–0.42 mmol/litre	Plasma or serum
Urea	15–40 mg/100 ml 2.5–6.6 mmol/litre	Plasma or serum

* Requires free flowing blood, so venepuncture should be performed without tourniquet or tourniquet should be removed before blood is withdrawn. If borderline high result is obtained, repeat determination with patient fasting at 10 in the morning.

TABLE 13. CHEMICAL PATHOLOGY: BLOOD—(cont.)

	<i>Normal range</i>	<i>Specimen required</i>
Glucose fasting	60–90 mg/100 ml	Anticoagulated (fluoride oxalate)
Sugar (reducing substances)	3–5 mmol/litre	whole blood
	65–110 mg/100 ml	Anticoagulated (fluoride oxalate) whole blood
Protein		
Total	6.0–7.4 g/100 ml 60–74 g/litre	Plasma or serum
Albumin	Dependent on posture	
	3.6–4.7 g/100 ml	Plasma or serum
	36–47 g/litre	
Fibrinogen	Dependent on posture	
Bilirubin	200–500 mg/100 ml	Plasma (oxalate)
Total		Plasma or serum
‘Direct reacting’	less than $\begin{cases} 1 \text{ mg/100 ml} \\ 17 \text{ } \mu\text{mol/litre} \end{cases}$	
	less than $\begin{cases} 0.2 \text{ mg/100 ml} \\ 3 \text{ } \mu\text{mol/litre} \end{cases}$	
Osmolality	283–295 mosm/kg	
Alkaline phosphatase	3–13 K.A. units	Plasma or serum
Acid phosphatase		
Total	0.5–5.0 K.A. units	
Formol-stable	0.5–4.5 K.A. units	Plasma freshly separated
Amylase		
Aspartate amino-transferase (AspAT) (GOT)	Units depend on laboratory method used	Plasma or serum
Lactate dehydrogenase (LDH)		
Creatinine phosphokinase		
Aldolase		

Carotenoids (as β -carotene)	50–200 $\mu\text{g}/100\text{ ml}$	Plasma or serum
Vitamin A	60–135 i.u./100 ml 0.7–1.7 $\mu\text{mol}/\text{litre}$	Plasma or serum
Cortisol (marked diurnal variation)	Midnight 1–10 $\mu\text{g}/100\text{ ml}$ 9.30 a.m. 5–25 $\mu\text{g}/100\text{ ml}$	Plasma (separated immediately)
Protein-bound iodine	4.0–8.0 $\mu\text{g}/100\text{ ml}$ (higher in women on the 'pill', on oestrogen therapy or in pregnancy)	Plasma or serum†
T 4	4.5–13 $\mu\text{g}/100\text{ ml}$	Plasma
T 3 (resin-binding test) uptake	92–117 %	Plasma
Effective thyroxine index (ETI)	4.5–13.2 %	Plasma
Lipids		
Cholesterol, total	140–300 $\text{mg}/100\text{ ml}$ 3.6–7.8 mmol/l	Plasma or serum
Triglycerides (as triolein)	25–150 $\text{mg}/100\text{ ml}$ 0.28–1.70 mmol/litre	Serum
Lipoprotein electrophoresis	Results reported according to techniques used	Serum
Protein electrophoresis		Serum

† Blood must *never* be taken for this purpose from patients who have recently received iodine containing X-ray contrast media. Such blood will spoil the apparatus used, until it has been thoroughly cleaned. Results are unreliable in patients taking iodine in any form (including dermatological preparations and patent chest or asthma preparations) and for 4 weeks after an excretion pyelogram, six months after an arteriogram and at least 5 years after a myelogram.

TABLE 13. CHEMICAL PATHOLOGY: URINE---(cont.)

	<i>Normal value</i>
Creatinine	0.7-2.0 g/24 hours 5.5-17 μ mol/24 hours
Urea	15-30 g/24 hours 250-500 mmol/24 hours
Uric acid	400-800 mg/24 hours 2.4-4.8 mmol/24 hours
Total proteins	10-15 mg/24 hours
Total porphyrins	0-300 μ g/24 hours
Coproporphyrin	0-250 μ g/24 hours
Amylase	4-32 Wohlgemuth units
Calcium	140-360 mg/24 hours 35-90 mmol/24 hours
Sodium	60-180 mmol/24 hours (markedly dependent on diet)
Potassium	50-100 mmol/24 hours (markedly dependent on diet)
Chloride	60-180 mmol/24 hours (markedly dependent on diet)
Phosphate	500-1500 mg/24 hours 16-48 mmol/24 hours (markedly dependent on diet)
Magnesium	80-120 mg/24 hours 3.3-4.9 mmol/24 hours

Copper

10-50 $\mu\text{g}/24$ hours
0.2-0.8 $\mu\text{mol}/24$ hours

Lead

30-80 $\mu\text{g}/24$ hours
0.14-0.39 $\mu\text{mol}/24$ hours

5-hydroxyindolylacetic acid (5-HIAA)
4-hydroxy-3-methoxy-mandelic acid (HMA, VMA)

less than 7 mg/24 hours
1-8 mg/24 hours

Steroids

Age 20-40 Males

Markedly dependent on age and sex

17-OS 5-20 mg/24 hours
17-OGS 7-18 mg/24 hours
17-OS 3-15 mg/24 hours
17-OGS 5-15 mg/24 hours

Females

Osmolality

Depends on diluting and concentrating function of kidneys:
usual range 100 to 1150 mosm/k

Cerebrospinal fluid

Total protein

(See p. 261)
10-40 mg/100 ml
0.1-0.4 g/l

glucose

4.5-90 mg/100 ml
2.5-5 mmol/l

Faecal fat

(See p. 64)
1-6 g/24 hours for single collection (24 hours) but not more
than 12 g in 3-day collection (72 hours)

APPENDIX

APPENDIX

SI UNITS

In this edition the *Système International d'Unités* has been used as far as possible. This system aims to derive all measurements from seven basic units and to express all measurements as decimal fractions or multiples of these. Of the seven basic units the four which appear in this book are:

<i>Physical quantity</i>	<i>Name of SI unit</i>	<i>Symbol</i>
length	metre	m
mass	kilogram	kg
time	second	s
amount of substance	mole	mol

and the prefixes indicating the decimal fractions and multiples are:

<i>Fraction</i>	<i>Prefix</i>	<i>Symbol</i>
10^{-1}	deci-	d
10^{-2}	centi-	c
10^{-3}	milli-	m
10^{-6}	micro-	μ
10^{-9}	nano-	n
10^{-12}	pico-	p
10^{-15}	femto-	f
<i>Multiple</i>	<i>Prefix</i>	<i>Symbol</i>
10	deca-	da
10^2	hecto-	h
10^3	kilo-	k
10^6	mega	M

The litre ($1 = \text{dm}^3$) is also recognized as the unit of volume.

It follows that when SI is adopted certain familiar terms will no longer be used, as is the case with measures of volume. A cubic centimetre (cc, cm^3) is replaced by the millilitre (ml) and the cubic millimetre (cmm, mm^3) by the microlitre (μl). In linear measure the micron (μ) should no longer be used; the correct unit is the micrometre (μm). Blood, intrauterine and intra-ocular pressures are measured in millimetres of mercury (mm Hg) and intrathecal pressures in centimetres of water (cm H_2O). It is recommended that the medical calorie or kilocalorie should now be converted to the joule ($1 \text{ kCal} = 4186.8 \text{ J}$).

Further information on SI units may be obtained from *The Use of SI Units*, Publication PD 5686 of the British Standards Institution, and useful information on the SI units commonly used in medicine and biology is available in *Units, Symbols and Abbreviations: A Guide for Biological and Medical Editors and Authors*, published by the Royal Society of Medicine.

SOME APPROXIMATE CONVERSION TABLES

1 fluid ounce (fl oz)	= 28 ml
1 gallon UK (gal)	= 4.5 litres
1 grain (do not abbreviate)	= 65 mg
1 inch (in)	= 25.4 mm
1 foot (ft)	= 0.3 m
1 ounce (oz)	= 28 g
1 pound (lb)	= 0.45 kg
1 calorie (cal)	= 4.2 J
1 kilocalorie (medical calorie)	= 4.2 kJ

CENTIGRADE AND FAHRENHEIT SCALES

The Centigrade (Celsius) scale is preferred.

The following table shows the relationship of the Centigrade and Fahrenheit scales, as far as is likely to be required in clinical work.

<i>Centigrade</i>	<i>Fahrenheit</i>	<i>Centigrade</i>	<i>Fahrenheit</i>
110	230	36.5	97.7
100	212	36	96.8
95	203	35.5	95.9
90	194	35	95
85	185	34	93.2
80	176	33	91.4
75	167	32	89.6
70	158	31	87.8
65	149	30	86
60	140	25	77
55	131	20	68
50	122	15	59
45	113	10	50
44	111.2	5	41
43	109.4	0	32
42	107.6	-5	23
41	105.8	-10	14
40.5	104.9	-15	5
40	104	-20	-4
39.5	103.1		
39	102.2	0.54	1
38.5	101.3	1	1.8
38	100.4	2	3.6
37.5	99.5	2.5	4.5
37	98.6		

To convert Fahrenheit to Centigrade:

$$X^{\circ}\text{F} - 32 \times \frac{5}{9} = Y^{\circ}\text{C}$$

To convert Centigrade to Fahrenheit:

$$X^{\circ}\text{C} \times \frac{9}{5} + 32 = Y^{\circ}\text{F}$$

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1969. 2nd ed. 446 pp. 104 illus.

Italian ed.: Il Pensiero Scientifico, Rome/Sinhala ed.: Ceylon Government/Spanish ed.: Editorial Paidos, Buenos Aires.

Cardiology

By D. G. JULIAN, MA, MD, FRCPE, MRACP, Consultant Physician, Royal Infirmary, Edinburgh; Senior Lecturer in Medicine, University of Edinburgh. Foreword by Sir STANLEY DAVIDSON, Emeritus Professor, University of Edinburgh.

This is the second edition of Dr. Julian's widely acclaimed volume which describes and explains with great clarity the manifestations of cardiac disease to the non-specialist. The framework of the book remains the same but detailed revision has been made throughout the text and additions have been made to the illustrations. *The Lancet's* reviewer wrote of the first edition: "This excellent short book will be of great help to undergraduate students. It clearly states the basic principles of modern cardiology, based on clinical, electrocardiographic, and haemodynamic findings . . . Dr. Julian's

volume is most strongly recommended, and its admirable presentation will bring much of value to the family doctor and to the general physician, as well as to those who are not yet qualified."

Contents. Incidence of Heart Disease – Myocardial Function: Heart Failure – Electrical Activity of the Heart – Disorders of Rate, Rhythm and Conduction – Symptoms of Heart Disease – Physical Signs of Heart Disease – Radiology and Allied Techniques of Investigation – Disease of the Coronary Arteries – Diseases of the Pericardium, Myocardium and Endocardium – Rheumatic Heart Disease – Disorders of the Cardiac Valves – Congenital Heart Disease – Hypertension and Heart Disease – Diseases of the Aorta – Disorders of the Lungs and Pulmonary Circulation – Systemic Disorders and the Heart – Psychological Aspects of Heart Disease – Surgery of the Heart – Index.

1973. 2nd ed. 335 pp. 105 illus.

Turkish ed.: University of Istanbul.

Dermatology

By E. LIPMAN COHEN, MA, MB, B CHIR (CAMB), Consultant Dermatologist to St. Margaret's Hospital, Epping, Princess Alexandra Hospital, Harlow and Oldchurch Hospital, Romford; and J. S. PEGUM, MA, MD (CAMB), FRCP, Physician to the Skin Department, The London Hospital; Consultant Dermatologist to the Queen Elizabeth Hospital for Children, London.

This newly revised edition gives the student a practical, up-to-date account of the knowledge he requires of diseases of the skin and their treatment, and provides the general practitioner with a guide to the latest developments in diagnosis and therapy. The revision has been extensive with some shortening of the text but with the inclusion of new material. A particular feature of this edition is the addition of eight plates of photographs and several new drawings. Dermatology is a rapidly changing subject – this book will enable both student and practitioner to keep abreast of current progress.

Contents. Biology of the Skin; Principles of Diagnosis and Treatment – Genetic and Developmental Abnormalities – Diseases; Bacterial, Viral, Fungous, Parasitic – Eczema; Contact Dermatitis;

Dermatoses due to Physical Agents – Drug Eruptions – Urticaria – Purpura – Vascular Disorders – ‘Autoimmune Diseases’ – Bullous Eruptions and Dermatitis Herpetiformis – Lichen Planus and Parapsoriasis – Tumours of the Skin – Diseases of Infancy and Childhood – Pruritus, Prurigo, and Self-inflicted Eruptions – Pityriasis Capitis; Seborrhoeic Dermatitis, Acne – The Hair, Nails and Cutaneous Manifestations of VD and Yaws – Index.

1970. 2nd ed. 222 pp. 14 illus. 8 plates.

Spanish ed.: Eudeba, Buenos Aires.

Embryology

By M. B. L. CRAIGMYLE, MB, CHB, MD, Senior Lecturer in Anatomy and Histology, University College, Cardiff.

Provides the medical student with a sound, basic knowledge of embryology which will elucidate the genesis of abnormal development and will pave the way for further study of pathology, immunology and clinical medicine.

‘Accurate, brief, and inexpensive.’ *Middlesex Hospital Journal*, reviewing the 1st edition.

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1975. 2nd ed. 256 pp. 105 illus.

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By I. A. D. BOUCHIER, MD, FRCP, Reader in Medicine, The Royal Free Hospital, London.

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1973. 302 pp. 17 illus.

Medical Microbiology

By C. G. A. THOMAS, MA, BM, BCH (OXON), MRCP (LOND), FC PATH, Consultant Bacteriologist, Norfolk and Norwich Hospital; Formerly Senior Lecturer in Clinical Pathology, Guy's Hospital Medical School, London.

A concise general account of microbiology with emphasis on those aspects which are relevant to the day-to-day practice of medicine. The book is based on lectures given to medical students at St. Thomas's Hospital, Guy's Hospital and the University of Rochester, New York, modified in the light of experience gained as a hospital bacteriologist in Norwich. Besides general revision for the new edition, line illustrations have been included in the text together with a new chapter on helminths.

Contents: Preface – Origins of Microbiology – General Properties of Bacteria – General Properties of Viruses – Cultivation of Organisms –

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1973. 3rd ed. 383 pp. 32 illus.

Spanish ed.: Editorial Paidós, Buenos Aires.

Obstetrics

By J. M. HOLMES, MD, BS (LOND), FRCOG, Consultant Obstetric and Gynaecological Surgeon, University College Hospital, London.

The second edition has been substantially enlarged to give a concise but comprehensive survey of modern obstetrics sufficient to meet the requirements of the MB and BS examinations. Several new chapters and many new illustrations have been introduced to cover topics such as drug dependence in pregnancy, placental function, placental insufficiency and foetal distress. Modern methods of assessing the foetus *in utero* are described, and there is more detail on maternal diseases in pregnancy and certain obstetric operations. Recommended drugs and dosages are listed with metric measurements throughout.

‘Many students need a concise textbook as an introduction to obstetrics and, perhaps more importantly, for last-minute revision before examinations. This manual meets these needs ideally.’ *British Medical Journal*.

Contents: Development of the Ovum – Anatomy and Physiology of the Foetus – Pregnancy: Changes in the Mother – Symptoms and Signs – General Management – Hydramnios – Abortion – Diseases Associated with Pregnancy – Displacement of the Pregnant Uterus – Vomiting – Toxaemia – Extra-Uterine Pregnancy – Antepartum Haemorrhage – Psychiatry in Childbearing – The Pelvis in Relation to Labour – Clinical Phenomena of Labour – Vertex Presentations –

Management of Natural Labour – Puerperium – Placental Insufficiency – Foetal Distress – Asphyxia Neonatorum – The Infant after Birth – Lactation – Malpresentations – Multiple Pregnancy – Presentation and Prolapse of the Cord – Prolonged Labour – Complications of the Puerperium – Puerperal Infection – X Rays and Ultrasonics – Induction of Labour – Version – Instrumental Delivery – Caesarean Section – Anaesthesia in Labour – Vital Statistics – Drugs Used in Obstetrics – Index.

1969. 2nd ed. 308 pp. 51 illus.

Ophthalmology

By KENNETH WYBAR, MD, CHM, DOMS, FRCS, Ophthalmic Surgeon, the Hospital for Sick Children, Great Ormond Street, the Royal Marsden Hospital; Surgeon, Moorfields Eye Hospital, London; Lecturer in Ophthalmology, University of London.

This book provides a factual account of the essential features of the practice of ophthalmology with particular regard for the medical student, and for the general practitioner who is concerned with the recognition of eye disease in its early stages, also for the specialist in other branches of medicine who is aware of the importance of an ophthalmic examination in an adequate assessment of many forms of systemic disease. However, the value of this comprehensive yet concise account of the subject has been appreciated by those working for a higher examination and even by the established ophthalmic surgeon. In the second edition therefore the author has included certain new features including the A and V phenomena, blow-out fracture of the orbit and the application of fluorescein angiography.

Contents. Basic Methods of Examination – Estimation of the Visual Acuity – Diseases of the Conjunctiva, the Cornea, the Sclera, the Uveal Tract, the Retina, the Optic Nerve, the Lens, the Vitreous Body, the Eyelids, the Lacrimal Apparatus – Disorders of the Extrinsic Ocular Muscles – Diseases of the Orbit – Glaucoma – The Afferent Visual Pathway – Injuries – Care of the Blind – Index.

1974. 2nd ed. 370 pp. 80 illus.

Spanish ed.: Editorial Paidós, Buenos Aires.

Paediatrics

By JOHN APLEY, CBE, MD, FRCP, Consultant Paediatrician, United Bristol Hospitals and Bath Clinical Area.

This book, a new title in the Series, by a distinguished paediatrician is primarily addressed to medical students but is also of great interest to general practitioners for it is original both in its manner and presentation of the subject. While dealing with the fundamentals of paediatrics, and deliberately emphasising the ways adult and childhood medicine are dissimilar, stress is laid on growth as growth influences disease and disease growth. Methods of examining children are taught throughout and therefore this book is an extension of cot-side and clinic teaching. Perhaps its most outstanding feature is the author's happy and personal way of bringing the reader into the discussion of the diagnostic and clinical problems of childhood medicine.

Contents. You and the Child in Hospital – Not Mini-Adults: The Chronology of Disease – Illness and Growth – What is Normal? Growth and Development – Diagnosis and Treatment – Skin Disorders – Respiratory Tract Disorders – Disorders of the Heart – Gastrointestinal Disorders – Disorders of the Nervous System – Disorders of Thinking and Speaking – Disorders of Emotion and of Behaviour – Endocrine Disorders – Disorders of Metabolism – Renal Tract Disorders – Disorders of Bones and Joints – Collagen and Muscle Diseases – Blood Disorders – The Newborn Infant and his Disorders – Nutrition and Feeding – 'It Runs in the Family' – Appendices – Index.

1973. 448 pp. 8 pp. plates, 30 illus.

Pathology

By J. R. TIGHE, MD, BSC, FRCPE, MRCP, MRC PATH, Surgical Pathologist, St. Thomas's Hospital and Medical School, London.

This is a radically revised and enlarged edition of the popular book originally written by the late Dr. J. R. Pinniger, Dr. Tighe has retained the original division into two parts, General Pathology and the Pathology of the Systems. Every chapter has had new material added and there are new chapters on allergy, auto-immunity and

collagen diseases, gene and chromosome pathology, and the central nervous system. An additional chapter deals with those skin diseases of importance to the student in his pathology course. Suggestions for further reading have been included after the preface and at the end of appropriate chapters. ✓

‘The author has fulfilled his aim, in producing a book that provides a useful readable introduction to the subject of pathology for the student about to start his clinical studies and also a compact account of the subject for the more senior student who is revising for the Part I of the Final MB.’ *St. Mary’s Hospital Gazette*.

Contents. Part I: Introduction – Inflammation and Infection – Cell Necrosis, Degenerations and Infiltrations – Circulatory Disturbances – Hypertrophy; Hyperplasia; Atrophy; Aplasia; Hypoplasia – Pathological Effects of Ionising Radiation – Protozoal and Helminthic Infections – Pathological Reactions to Virus Infections – Pathology of the Fluid Compartments – Allergy; Anaphylaxis; Auto-immunity; Collagen Diseases – Gene and Chromosome Pathology – Neoplasms (New Growths) – The Aetiology of Cancer. *Part 2:* Pathology of the Systems: Circulatory, Respiratory, Tissues of Head and Neck, Alimentary System, Urinary Tract, Male Genital Tract, Female Genital Tract, Breast, Thyroid, Parathyroids, Adrenals and Pituitary; Diseases of the Haemopoietic and Allied Systems: The Skeletal System; Central Nervous System; The Skin – Index.

1972. 3rd ed. 319 pp.

Pharmacology

By R. G. PENN, MB, BCH (WALES), Lecturer in Pharmacology, Charing Cross Hospital Medical School, London.

This provides an account of those aspects of pharmacology that are important to the medical student starting the subject and later applying it in his clinical years. The basic scientific aspects are emphasised throughout and this book therefore provides the reader with a sound foundation of knowledge that will stand him in good stead throughout his career. Drugs have been grouped as far as possible under the body system they mainly affect, but dosages are not given since these must vary according to the needs of the patient.

'As a concise introduction for the preclinical student the book succeeds. It contains all the necessary information for 2nd MB and provides a good understanding for later reference.' *University College Hospital Gazette*.

Contents. General Pharmacology – The Autonomic Nervous System – Voluntary Muscle and the Neuromuscular Junction – Smooth Muscle – The Endocrine System – The Vitamins – The Circulation – The Blood – The Kidney – The Central Nervous System – Local Anaesthetics – The Alimentary Tract – Chemotherapy – Antibacterial, Antifungal and Antiviral Drugs – Antiprotozoal, Anthelmintic and Other Antiparasitic Drugs – Cytotoxic Drugs – Immunity and Immunological Products – Antiseptics and Disinfectants – Index.

1974. 290 pp. 15 illus.

Spanish ed.: Eudeba, Buenos Aires

Preventive Medicine, Community Health and Social Services

By J. B. MEREDITH DAVIES, MD, DPH, Director of Personal Health and Social Services, City of Liverpool, Lecturer in (Preventive) Paediatrics, University of Liverpool.

The present structure of medical care is in a state of transition, and important changes have already taken place since this book was first published in 1966. The author describes in this second edition the present state of preventive medicine, community health services and community social services in the United Kingdom.

With the advance towards full community care of the patient and coordination of all medical and para-medical services, this up-to-date and succinct account of these services is particularly opportune. Medical students, general practitioners and all who are training to serve in the field of community care will find this book an invaluable guide.

Contents. Preventive Medicine; History – Vital Statistics – Epidemiology; Infectious Diseases, Airborne or Droplet Infections, Faecal-Borne Infections, Other Infectious Diseases, Prevention of

non-Infectious Disease – Prevention of Disease by Immunisation – Preventive Medicine in General Practice – Care of the Worker – Food and Nutrition – International Health Control; Community Health Services; Administration of the Health Services – Maternity Services – Child Health Services – Health of the School Child – Health, Education and Other Community Health Services – Environmental Health Control – Pests and Parasites – Administration of the Social Services – Care of the Elderly – Care and Rehabilitation of the Handicapped – Care of the Mentally Disordered – Care of Children in Need – Home Help Services and Care of Homeless Families – Index.

1975. 3rd ed. 342 pp. 6 illus.

Psychiatry

By E. W. ANDERSON, MD, MSC, FRCP, FRC PSYCH, Emeritus Professor of Psychiatry, University of Manchester; Lord Chancellor's Visitor; revised by W. H. TRETHOWAN, MA, MB (CANTAB), FRCP, FRACP, FRC PSYCH, (HON) FANZCP, Professor of Psychiatry, University of Birmingham.

The new edition of a concise account of current practical psychiatry for use not only by medical students but also by those in whose training a knowledge of psychiatry is required, such as social workers. Previous editions have also proved to be useful and acceptable textbooks for doctors studying for the Diploma of Psychological Medicine.

Psychiatry, while continuing to encompass a growing number of related disciplines, still rests firmly upon clinical observation rather than upon theoretical speculations. Professor Trethowan has made a thorough revision of the entire text while maintaining the basic orientation of Professor Anderson's book.

Contents. Introduction – Psychopathology – Psychiatric Examination – Aetiology and Classification – Organic Disorders; Delirious and other Acute States, Chronic Brain Syndromes – Alcoholism and Drug Dependence – Epilepsy – Schizophrenia – Paranoid States – Affective Disorders – Personality Disorders – Neurotic Reactions – Anxiety States – Hysteria – Obsessive-Compulsive Disorders – Sexual Anomalies and Perversions – Psychiatry and General Medicine –

Child Psychiatry – Mental Subnormality – Treatment; Psychological and Social, Physical – Social Aspects – Legal Considerations – Index.

1973. 3rd ed. 375 pp.

Lists for Further Reading are included in all the latest editions. Each Concise Medical Textbook is 187 mm. × 127 mm., and bound in strong limp cloth.

Also available:

Stewart/Bacteriology and Immunology for Students of Medicine 'Formerly Bigger's Handbook of Bacteriology'

By F. S. STEWART, MD, FRCPI, FC PATH, MRIA, Professor of Bacteriology and Preventive Medicine, University of Dublin; Consultant Bacteriologist, Sir Patrick Dun's, Adelaide, Meath and Royal City of Dublin Hospitals; Consultant Serologist, Rotunda Hospital, Dublin.

This standard textbook presents the fundamentals of medical microbiology and immunology in a way designed specifically to meet the needs of the medical student.

Contents. The General Properties of Bacteria – Microscopic Examination of Bacteria – The Cultivation of Bacteria – Disinfection and Sterilisation – Bacteria in Health and Disease – Antigens and Antibodies – Antigen-Antibody Reactions – Immunity – Hypersensitivity – Antibiotics and Other Antibacterial Agents Used in Chemotherapy – Diagnostic Bacteriology – The Classification of Bacteria – The Staphylococci – The Streptococci – Pneumococci – The Neisseriae – Bacillus – The Corynebacteria – Commensal Intestinal Bacteria – The Salmonellae – The Shigellae – Vibrio; Spirillum; Pseudomonas – The Pasteurellae – Haemophilus; Bordetella; Moraxella – The Brucellae – The Mycobacteria – Actinomycetes; Actinobacillus – L Forms; Mycoplasma; Streptobacillus; Listeria, Erysipelothrix, Bartonella – The Clostridia – Spirochaetes – The General Properties of Viruses – The Picornaviruses – Arboviruses – Rabies and Miscellaneous CNS Viruses – The Poxviruses – The Myxoviruses – The Herpesviruses – Hepatitis – Miscellaneous Viruses – Chlamydiae – The Rickettsiae – Bacteriophages – The Pathogenic Fungi – Appendix – Bibliography – Index.

1968. 9th ed. 248 mm. × 159 mm. 610 pp. 76 illus. 3 col. plates.

And:

Merskey and Tonge/Psychiatric Illness

By H. MERSKEY, MA, DM, FRC PSYCH, Physician in Psychological Medicine at the National Hospitals for Nervous Diseases, London; and W. L. TONGE, MD, FRC PSYCH, Consultant Psychiatrist, United Sheffield Hospitals. Honorary Lecturer in Clinical Psychiatry, University of Sheffield.

Some 20 per cent of the patients in general practice are known to present with psychiatric problems and only a few of them can be referred to the specialist. The special purpose of this book is to provide an introduction to psychiatry through which practitioners and students can resolve some of these problems. The authors examine the symptoms which the patient presents as well as incorporate sufficient detail to cover the major types of psychological illness adequately for the general practitioner. While their views on the role and use of psychotherapy have developed since the first edition, they continue to show how psychotherapy operates in practical day-to-day consultations.

In the second edition, the sections on psychosomatic illness, subnormality, deviant behaviour and senile diseases have been revised or enlarged. The sections on hypnosis, psychotherapy, behaviour therapy, marital problems, children, and drug treatment have been rewritten and brought up to date. The chapter on Child Psychiatry is written by Dr. R. A. Bugler, Consultant in Child and Adolescent Psychiatry, North Derbyshire.

Contents. Part I. Introductory Section: The Subject Matter of Psychiatry – The Doctor's Problems – Examining the Patient – Part II. General Problems in Consultation: Common Symptoms – Neurosis – Social Factors – Functional Psychoses – Physical Illness and Psychiatric Disorder – General Principles of Treatment – Part III. Special Problems in Consultation: Psychosomatic Illness – Children and Adolescents – Emotional and Sexual Disorders in Marriage – Old Age – Loss of Memory – Subnormality – Deviations of Personality and Behaviour – Compensation Cases – Part IV. Psychiatric Emergencies – The Acutely Disturbed Patient – Suicide and Attempted Suicide – Appendix on Drug Treatment – Index.

1974. 2nd ed. 216 mm × 128 mm. 304 pp.

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Baillière's Medical Books

New editions may have been published since this list was printed; details quoted do not necessarily refer to current editions, and prices may have been increased.

This is the kind of book that every student requires for it deals with the most important of the many skills required by the general practitioner - that of accurate and comprehensive clinical examination.

HUTCHISON'S CLINICAL METHODS is full of practical information on the fundamental questions to ask, how to examine the patient, and what investigations to request in order to arrive at the correct diagnosis. It therefore serves the student beginning his clinical experience, and will prove a handy and reliable reference in his postgraduate years.

Much of the text has been rewritten for this edition and many of the illustrations have been replaced. Metric and SI units are used throughout. The early part of the book has been rewritten, as have the chapters on the nervous system, the urine, and laboratory work. All remaining chapters have been carefully revised and brought up to date. There are 10 new plates and a number of new figures in the text.

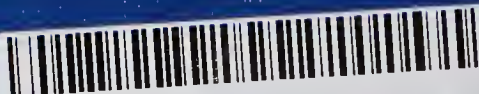
Reviews of the previous edition

'This is an excellent book for the student starting his clinical course. Read it and you won't go far wrong.'

Middlesex Hospital Gazette

'As good as ever and will continue to be of value to students and practitioners alike.'

East African Medical Journal



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